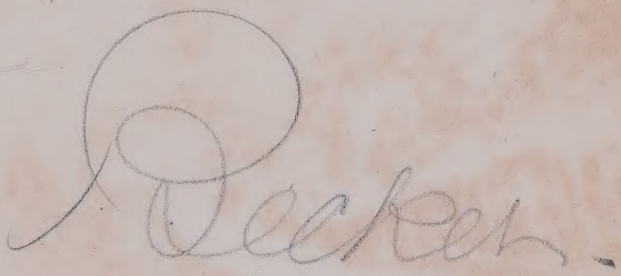

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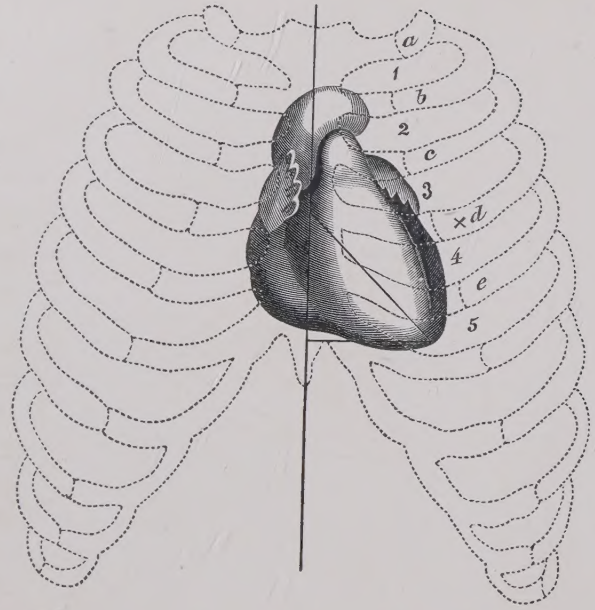
FIG. 1.



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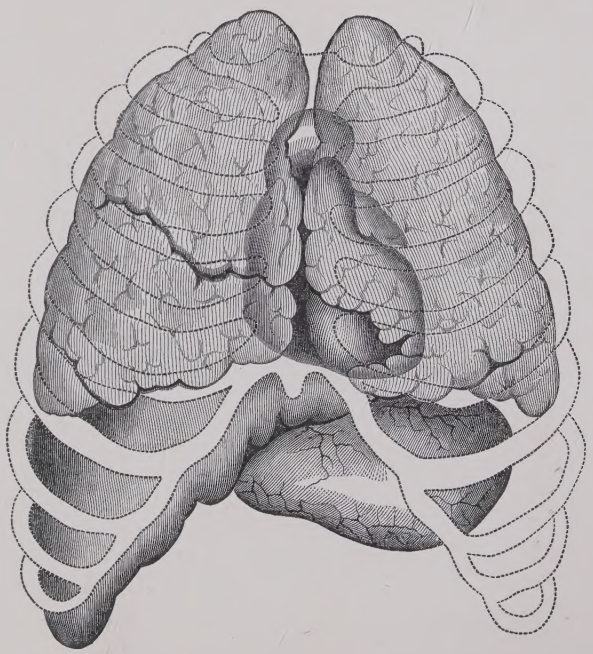

FOR DESCRIPTION, SEE PAGE XV.

D. L. ROKER,
11 COLLEGE ST.
PROV. R. I.

A

PRACTICAL TREATISE

ON THE

DIAGNOSIS, PATHOLOGY, AND TREATMENT

OF

DISEASES OF THE HEART.

BY

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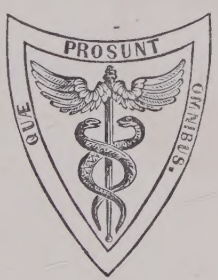

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TO

PROFESSORS HENRY MILLER, SAMUEL D. GROSS,

LUNSFORD P. YANDELL, LEWIS ROGERS, BENJAMIN R. PALMER,

BENJAMIN SILLIMAN, JR., J. LAWRENCE SMITH,

AND

T. G. RICHARDSON,

WITH WHOM

THE AUTHOR WAS FORMERLY ASSOCIATED

IN THE

UNIVERSITY OF LOUISVILLE,

This Volume

IS RESPECTFULLY DEDICATED.

PREFACE.

In the preparation of this volume, the aim has been to meet the wants of the medical student and practitioner by the production of a work devoted exclusively to diseases of the heart, and treating concisely, but comprehensively, of these diseases with reference to their diagnosis, pathology and treatment. Such a work, if satisfactorily executed, it is believed, can hardly fail to prove acceptable, in view of the importance of this class of diseases, the progress made in their investigation during the last few years, and the absence of any extended text-book, published in this country, having the same scope and objects, since the appearance of Dr. Hope's treatise twenty years ago. The need of a practical work on diseases of the heart is so apparent, that the present effort requires no apology; and if not successful, the fault must be imputed to the performance rather than to the undertaking. The author ventures to hope, in submitting this volume to the profession, that it may be found, in some measure at least, to supply a desideratum, the existence of which must have been felt by many practising physicians, and, more especially, by medical teachers and their pupils.

It will be observed that the arrangement of subjects in this work differs from that generally adopted. As regards the order in which the different diseases are considered, the plan usually pursued may be said to be synthetical, inflammatory affections being taken up first, and afterward the lesions which are, to a considerable extent, results of inflammation. A method which may be distinguished as analytical, has appeared to the author preferable. Pursuing this method, the work commences with the consideration of organic affections. Enlargement of the heart, occurring often consecutively to other lesions, takes precedence. To this subject the first chapter is devoted. Lesions affecting the walls of the heart naturally come next in order. These constitute the subject of the second chapter. Valvular lesions are then considered, occupying two chapters, and

a chapter is devoted to congenital malformations. Several affections which are incidental to diseases of the heart, are treated of in a distinct chapter. Then follow the inflammatory affections, and, afterward, functional disorders of the heart, three chapters being allotted to these classes of disease. Finally, thoracic aneurisms, which claim consideration in connection with diseases of the heart, are made the subject of the concluding chapter.

In writing the book, the end which the author has kept steadily in view is, a fair and full exposition of our present knowledge of the diagnosis, pathology and treatment of diseases of the heart. Recognizing clinical study as the great source of this knowledge, he has endeavored to make the cases reported by trustworthy observers, together with his own recorded experience, the basis of the work. Having long been in the habit of making records at the bedside, and having given for several years particular attention to diseases of the heart, he has accumulated notes of about two hundred cases of the various cardiac affections. The results of an analysis of these cases have been before him during the composition of the work. As a preliminary step, also, over one hundred fatal cases gathered from different authors, chiefly from the works of Hope, Stokes, Andry and Blakiston, were subjected to similar analysis. On the data thus obtained have been based, in a great measure, the statements and opinions which the work contains, endeavoring, however, not to introduce details and statistics to an extent to prove repulsive or fatiguing to the reader. But although it may be claimed in behalf of the work that it is something more than a compilation, not to have studied closely the literature of the subject, would have been an injustice alike to it, and to those by whose labors this department of practical medicine owes its present development. Of the authors to whom acknowledgments are due, the names of Bouillaud, Hope, Stokes, Walshe, Andry, Forget and Bellingham are to be especially mentioned. References to these and others will frequently occur in the following pages. The author has aimed to prepare a practical treatise, and he has therefore avoided, or dismissed with as much brevity as possible, speculative opinions and mooted questions involving discussions which would occupy space to the disparagement of matters relating more directly to medical practice. It may seem, nevertheless, to some, that the volume is out of proportion to the field of practical medicine to which it is restricted; but it is hoped there will be no reason to complain of a redundancy either in style or matter, and that the

reader will be led to attribute the size of the book to the progress of knowledge pertaining to diseases of the heart, together with their intrinsic claims on the attention of the student and practitioner.

A liberal share of the work is devoted to physical signs. But a just estimate of their practical importance will obviate any objection on this score. It is mainly owing to physical exploration that the study of these diseases has been prosecuted within the past few years with such remarkable success. Here, as in other classes of affections, the knowledge to be derived from clinical observation is increased in proportion to improvement in diagnosis, and it is evident that diseases cannot be judiciously treated unless correctly discriminated. The discrimination of diseases is confessedly the portion of our art which involves the most difficulty and calls for the greatest amount of skill. Hence, it is especially under this practical aspect that diseases in general claim careful and extended consideration. This remark, certainly, is not less applicable to diseases of the heart than to other nosological divisions. And the diagnosis of cardiac diseases is for the most part based on the physical signs. It is, therefore, by no means solely because these are interesting, but on account of their great practical importance, that so much space has been accorded to them in the present treatise. In treating of the physical signs, it was necessary to introduce some matter belonging properly to anatomy and physiology, viz., the relations of the heart to the walls of the chest and the adjacent viscera, the movements of the organ, and the normal heart-sounds. With reference to the movements and sounds of the heart, the author has been led by examinations of the healthy chest to conclusions which appear to have important practical bearings. The abnormal modifications of the heart-sounds have hitherto scarcely received sufficient attention. More importance is attached to them as diagnostic signs, and they are considered more fully in this work than in any other on the diseases of the heart with which the author is acquainted. As regards the sounds of the heart in health and disease, some original views are introduced, which have entered into a previous publication.[1]

In thus setting forth, briefly, the plan and objects of the work, the author assumes only to have spared no pains to render it

[1] On the Clinical Study of the Heart-Sounds in Health and Disease.—Transactions of the American Medical Association for 1858.

acceptable to the profession. All who have engaged in similar undertakings amidst the cares and distractions of active medical practice, will appreciate the difficulty of the task. But the time and labor which the author has bestowed upon it, will be more than requited by the approval of his medical brethren; and he is encouraged to hope for this reward by the favor with which his previous contributions to practical medicine have been received.

The author would express his thanks to Prof. John C. Dalton, Jr., for the two illustrations which form the frontispiece, and for other friendly offices; also, to Dr. Austin W. Nichols, formerly assistant to the chair of clinical medicine in the University of Buffalo, for his valuable assistance in collecting materials for the preparation of the work.

New York, *September*, 1859.

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DESCRIPTION OF THE PLATE

IN FRONT OF THE TITLE.

Fig. 1 illustrates the relations of the heart to the thoracic parietes. The letters *a*, *b*, *c*, etc., indicate the ribs. The figures 1, 2, 3, etc., mark the intercostal spaces. The vertical line denotes the median line. The right angled triangle extending over a portion of the surface of the heart, represents the "superficial cardiac region" as delineated on the chest with sufficient accuracy for practical purposes. The cross on the fourth rib shows the situation of the nipple. The relations of the ventricles, auricles, apex of the heart, aorta, and pulmonary artery, to the ribs and intercostal spaces, the median line and the nipple, are accurately indicated.

Fig. 2 illustrates the relations of the heart to the pulmonary organs, liver, and stomach. The quadrangular space in which the heart is uncovered by lung is the "superficial cardiac region," represented more accurately than in Fig. 1. The relative situations of the left lobe of the liver, the stomach, and inferior border of the heart, are correctly represented.

DISEASES OF THE HEART.

CHAPTER I.

ENLARGEMENT OF THE HEART.

DEFINITION and varieties of hypertrophy and dilatation—Normal dimensions and weight of heart—Enlargement by hypertrophy—Concentric hypertrophy—Symptoms and pathological effects of hypertrophy—Physical signs and diagnosis of enlargement and hypertrophy—Situation and anatomical relations of the heart in health—Alterations in degree and extent of dulness on percussion in hypertrophy—Altered situation and extent of the apex-beat, and abnormal force of impulse in hypertrophy, as determined by palpation—Mechanism of the heart's impulse—Abnormal modifications of the heart-sounds—Diminished extent and degree of the respiratory murmur and vocal resonance within the præcordia in hypertrophy, as determined by auscultation—Results of the clinical study of the heart-sounds in health—Enlargement of the præcordia and abnormal movements in hypertrophy, as determined by inspection—Increased size of the chest, as determined by mensuration—Summary of the physical signs of enlargement of the heart—Summary of the physical signs distinctive of enlargement by hypertrophy—Treatment of hypertrophy—Enlargement by dilatation—Symptoms and pathological effects of dilatation—Physical signs and diagnosis of dilatation—Summary of the physical signs distinctive of enlargement by dilatation—Treatment of dilatation.

ENLARGEMENT OF THE HEART is a term which embraces abnormal increase in the volume of this organ, in its weight, or, as is commonly the case, increase both in weight and volume. Augmentation of the volume of the heart, and of its weight, gives rise to different forms of enlargement, which, although usually associated, may exist each independently of the other. The heart may exceed the limits of health as regards weight, in consequence of an increased thickness of its walls, the normal bulk being retained. This may and does occur, although, in the vast majority of the cases in which the weight is augmented, the volume exceeds the healthy limits. On the other hand, the bulk of the heart may be abnormally great, the cavities being enlarged, and the thickness of the walls so far diminished, that the normal weight is retained. This form of

enlargement is also of very rare occurrence, the organ generally increasing in weight when its bulk is greater than in health. Abnormal increase of the heart in weight, due to morbid thickness of the walls of the organ, constitutes the condition called *hypertrophy*. Abnormal increase of the heart in volume, due to the morbid size of its cavities, constitutes the condition called *dilatation*.

These names, hypertrophy and dilatation, thus denote different forms of enlargement of the heart, presented sometimes separately, but usually together. Each of these two forms of enlargement are subdivided by writers into several varieties, the subdivisions being based on well-marked and important distinctions. Hypertrophy differs in different cases, according to the condition of the cavities, as regards size, associated with it. It exists in some cases without any alteration of the cavities, the latter remaining normal. This variety is called *pure* or *simple* hypertrophy. The cavities may be diminished in size below the limits of health. This must be admitted as a variety of hypertrophy, although its existence is denied by some. It has been distinguished as *concentric hypertrophy*, or *hypertrophy with contraction*. The variety occurring much more frequently than the others, in fact, that which exists in the vast majority of the cases in which the heart is hypertrophied, is characterized by the coexistence of dilatation to a greater or less extent. This variety is called *eccentric hypertrophy*, or *hypertrophy with dilatation*. The other form of enlargement, viz., dilatation, differs in different cases, according to the condition, as regards thickness, of the walls of the heart. Dilatation exists in some cases, the walls retaining their normal thickness. This is called *pure* or *simple* dilatation. It is obvious, however, that, in proportion to the dilatation, the heart is hypertrophied, assuming the walls to preserve their normal thickness, inasmuch as the mass of muscular structure and the weight of the organ under these circumstances must be increased. In other cases in which the capacity of the cavities is increased, the thickness of the walls is diminished. In this variety, the weight of the heart may not exceed, and may even fall below, that of health. This is distinguished as *dilatation with attenuated walls*, or *attenuated dilatation*.[1] The third variety of dilatation occurs with far greater frequency than either of the other

[1] In the rare instances in which the walls are so attenuated that the weight of the heart falls below the limits of health, the condition is one of atrophy. There is, however, no practical advantage in constituting this a distinct variety of enlargement.

varieties, and is characterized by the coexistence of hypertrophy, well marked, the dilatation, however, being predominant.

These subdivisions, although based on distinctions which are real and important, are somewhat complicated and embarrassing to the student. They are consistent with the different morbid conditions of the heart, as determined by examinations after death; but they are not accompanied by diagnostic criteria, by means of which they may always be discriminated at the bedside during life. A simpler arrangement is clinically more available, and suffices for all practical purposes. We may distribute all cases of enlargement of the heart into two classes, viz., 1st. Enlargement by hypertrophy; and 2d. Enlargement by dilatation. These classes will include, respectively, cases in which the hypertrophy and the dilatation are either simple or predominant. In cases of "enlargement by hypertrophy," the cavities may or may not exceed their normal capacity. Cases in which the cavities are diminished will also fall in this class. If the hypertrophy be neither simple nor *concentric*, it is included in this class whenever it is proportionately greater than the coexisting dilatation. The symptoms and signs enable the diagnostician to determine, often with positiveness, the existence of hypertrophy, which is either simple, or predominant over a coexisting dilatation; but to discriminate between the cases in which the hypertrophy is simple and those in which it predominates over coexisting dilatation, is a problem in diagnosis by no means easily solved. So in cases of "enlargement by dilatation," the amount of muscular structure may or may not exceed the limits of health. The diagnostic criteria of predominant dilatation are often sufficiently positive; but it is far less easy to decide whether the dilatation be accompanied with hypertrophy or attenuation. Moreover, as regards prognosis and treatment, after the existence and degree of enlargement are ascertained, it is enough to determine which form of enlargement predominates, hypertrophy or dilatation. In treating of enlargement of the heart, I shall follow the simple classification just indicated.

As a point of departure for the study of those affections of the heart which consist of abnormal deviations in size, its normal dimensions and weight are to be considered. The healthy standards in these respects are obtained by measuring and weighing a sufficiently large number of hearts presumed to be devoid of disease. As regards measurements, the diameters and the thickness of the walls are the points which have reference to the affections to be

treated of in this chapter. The dimensions of the orifices and valves will be considered in connection with lesions in this situation. The researches of Bizot and others show that the volume of the heart varies according to sex and age. It is somewhat greater in the male than in the female, and it increases slowly, but progressively, from infancy to old age. It is to be observed that diametrical measurements after death are liable to be affected by incidental circumstances, by which they are rendered only approximatively correct. The degree of contraction varies according to the quantity of blood which the cavities contain at the time of death. Observations show that when death occurs from hemorrhage and from diseases attended by rapid loss of fluids, the cavities are much diminished, and the volume proportionately small; while, on the other hand, if the cavities are distended with blood, they are dilated, and the volume increased in proportion. In consequence of these variations, the measurements of the entire organ, made by means of careful percussion and auscultation during life, are as reliable, if not more so, than those made in the dead subject. In Bizot's tables are exhibited the mean measurements of the length, breadth, and depth of the heart as a whole, and of the two ventricles, respectively, in the two sexes at different ages. As standards for comparison, with reference to the existence of abnormal enlargement, it is sufficient to take into view the vertical and transverse diameters, the contents of the cavities having been removed. And it suffices to express the normal averages in figures approximating to the exact results obtained by taking the mean of measurements, disregarding fractional amounts, which the student cannot be expected to remember. Moreover, the results obtained by different observers present considerable variation, which, in view of the facts just stated, might be expected. Adopting, as a basis, the measurements by Bizot and others, it is sufficiently exact to say that the average length of the heart, measured from apex to base on its anterior surface, in the male, between the ages of thirty and fifty, is about four inches, being in the female somewhat less; and that the width, measured at its widest part, in the male, is a small fraction over four inches, being somewhat less in the female.[1]

[1] Farther details with regard to measurements of volume are dispensed with as practically not important in this connection. Bizot's extensive and elaborate researches, which will be again referred to, were published in the *Mémoires de la Société Médicale d'Observation de Paris*, 1836. For a summary of his results relating to the above points and others, the reader is referred to Hope on *Diseases*

The general remarks just made with reference to the normal volume of the heart, are also applicable to the thickness of the walls; the thickness is greater, as a rule, in males than in females, and it increases with age. It varies, also, according to the contraction of the heart at the time of death, dependent on the amount of blood contained within the cavities, and other circumstances. Hence, measurements here, as with respect to the diameters, in a collection of hearts, furnish results which are only approximations to correctness. Pursuing the same course as in expressing the normal standard of volume, it is approaching near enough to exactness to say that the wall of the left ventricle, at its thickest portion, in middle life, is not far from half an inch in the male, and in the female a fraction less. The thickest part of this ventricle is near its centre. The thickness is less near the base, and still less at the apex. The wall of the right ventricle, at its thickest portion, is a little over one-sixth of an inch, in the male, and in the female somewhat less. The thickest part of this ventricle is near the base, and the thinnest near the apex. The relative thickness of the two ventricles is, thus, in the ratio of 3 to 1. The average thickness of the right auricle is estimated to be about a twelfth of an inch, and of the left auricle somewhat greater.

The average normal dimensions of the heart as a whole, and of different parts of the organ, are important as standards of comparison by which to estimate abnormal changes. Their importance in this respect, however, is less than might, at first view, be imagined. The deviations from these standards, which are embraced within the limits of health, are to be taken into account. The range of normal variation, as regards the volume of the heart and thickness of its walls, is considerable. An addition of an inch or more to the vertical and transverse diameters may not be abnormal. So, a proportionate amount of increased thickness of the walls of the ventricles may be within healthy limits. To determine the line of demarcation between normal and abnormal deviations, is more difficult than to ascertain averages. It is not easy to fix a maximum and a minimum, beyond which the condition is always

of the Heart, Am. ed., edited by Pennock; to the work by Dr. Stokes on *Diseases of the Heart and Aorta;* and to Bellingham on *Diseases of the Heart*, Part I., Dub. ed. For results of measurements by Ranking, Gross, and others, Gross's *Path. Anat.*, third edition, and Dunglison's *Physiology*, eighth edition, may be consulted; see also *Traité Clinique des Maladies du Cœur*, par J. Bouillaud, which contains measurements by himself and strictures on the researches of Bizot.

morbid. And even were the boundaries definitely fixed, it might still be a matter of doubt in some individual cases in which the limits were not exceeded, whether the condition was not abnormal. Enlargement of the heart sufficient to be of much pathological importance, is generally so well marked that its existence does not admit of doubt. Practically, therefore, the want of precise data for defining rigorously the confines of morbid anatomy, does not lead to serious inconvenience. These remarks are applicable, not only to the dimensions of the heart already considered, but, equally, to the capacity of its cavities and to its weight.

The cavities of the heart are not readily measured. Their capacity varies, irrespective of intrinsic normal differences, according to the quantity of blood which they contain, and the condition of the muscular walls at the time of death. They are also affected by *post-mortem* changes. The two ventricles and auricles do not, in health, present any marked disparity in capacity. The right ventricular and auricular cavities are somewhat larger than the left. This is probably in some measure due to the greater distension of the former, in consequence of the larger accumulation of blood at the time of death; but, aside from this circumstance, observations show that some disparity exists. The capacity of the auricles is somewhat greater than that of the ventricles. In order to represent the average size of the cavities, it has been the custom to say that each will contain a hen's egg of medium size. This homely illustration is sufficiently exact. The right ventricle is estimated to contain about two ounces of liquid, and the left ventricle not much over an ounce and a half. Dilatation, when it exists to an extent to constitute a lesion of much importance, and as it is met with in autopsies of subjects dead with cardiac disease, is usually sufficiently well marked to be recognized and its degree determined by the eye.

The average weight of the heart, as determined by weighing a large number presumed to be free from disease, and taking the mean, is not easily fixed with precision, because the results in different hands differ considerably, a fact which goes to show that the variations within the limits of health are considerable. For the reasons, however, which were stated with respect to the average size of the organ, mathematical exactness in giving the average weight is not practically important. The range of normal variation is more important to be considered. Bouillaud, from the results of weighing the hearts in thirteen subjects, fixed the average

weight, between the twenty-fifth and sixtieth years, at from eight to nine ounces. Dr. Clendinning weighed a much larger number, nearly four hundred, all from subjects over puberty, and the mean was about nine ounces.[1] Dr. John Reid found the average of eighty-nine male hearts to be a little over 11 ounces, and of fifty-three female hearts a little over nine ounces.[2] It is sufficient to say that the average weight is between eight and ten ounces. And it is to be borne in mind that if it be found to exceed this average, or fall below it by one and two ounces, it is by no means to be inferred that the condition is abnormal. The medium weight in the female is somewhat less than in the male. The weight, as well as the dimensions of the heart, also increases progressively up to an advanced period of life.

ENLARGEMENT BY HYPERTROPHY.

Under this title are included not only the rare instances in which the enlargement is due exclusively to increased thickness of the muscular walls, but all cases in which the hypertrophy, although associated with more or less dilatation, preponderates over the latter. In examining the heart, after laying open the cavities and removing their contents, the predominance of either hypertrophy or dilatation is generally obvious to the eye. The two forms of enlargement are combined, in different cases, in every degree of relative proportion. The question is, which contributes most to the morbid size, increase of the structure, or of the capacity of the cavities. Instances, however, occur in which these two elements of enlargement are about evenly balanced. On measuring and weighing the organ, the excess of weight is greater than the abnormal dimensions in proportion as the hypertrophy preponderates. The walls are more solid and resisting; the rounded form of the ventricles is retained when the organ is placed on its posterior surface, not being flattened by the collapse of the ventricular walls. If the increased thickness of the walls of the ventricles be due to *true* hypertrophy, they present externally, and on section, the appearances of healthy muscular structure. The microscope shows

[1] Avoirdupois weight in all the instances cited.

[2] Dunglison's Physiology.

the tissue to be normal. The deposit of fat upon the heart, between the fibres, with fatty degeneration of the latter, in some cases adds to the bulk, and gives rise to abnormal appearances of the surface and muscular substance. This constitutes a species of *false* hypertrophy, which affects certain of the symptomatic phenomena referable to the heart's action. In true hypertrophy, inasmuch as the size of the muscular fibrillæ is not increased, it follows that there takes place an actual hypergenesis of the tissue. The several portions of the heart may all participate in the enlargement, or it may be confined to one or more of the anatomical divisions without extending to the whole organ. In the great majority of cases all portions are involved, but they are rarely affected equally; the enlargement is more marked in some divisions than in others. The different portions may not present the same form of enlargement. Hypertrophy may predominate in one part and dilatation in another. This fact renders it necessary to consider the divisions of the organ separately. Of these divisions the left ventricle is the one most apt to be affected alone, and the enlargement is often relatively more marked here than in other portions, when the whole organ is more or less affected. An effect of enlargement is to alter the form of the organ. The width is increased more than the length. The heart is rendered abnormally broad and globular. The conoidal appearance is less marked than in health, the lower extremity being blunted, and the pointed apex, as it were, absorbed into the ventricle. This is more marked when the enlargement is confined to, or involves the right ventricle. Certain points relating to physical signs are explained by the change which the apex undergoes. The degree of hypertrophy varies greatly in different cases. The thickness of the left ventricle may be increased to an inch, an inch and a half, and even two inches. The walls of the other compartments may, in like manner, be doubled, tripled, and even quadrupled. The vertical and transverse dimensions may be five or six inches, or more. The weight may exceed two, three, four, and even five times the normal average. The largest weight among the instances that have come under my personal knowledge is forty ounces.

The pathological process giving rise to true hypertrophy is hyper-nutrition. With few, if any, exceptions, this process is a result of undue exercise of the muscular power of the organ. Pathologically considered, it is difficult to account for the production of muscular hypertrophy of the heart, except as a consequence

of some anterior abnormal condition which has induced, for a considerable period, augmented muscular power. The principle is the same as in the familiar examples of voluntary muscles becoming disproportionately developed when inordinately exercised. Clinical observation shows that in the majority of cases of hypertrophy, prior abnormal conditions do exist, which stand in a causative relation to this affection. The practical bearing of this view of the pathology is important. In much the larger proportion of cases of hypertrophy, the anterior causative conditions are obvious, and are seated in the heart itself, or in the large vessels. The affection in these cases may be distinguished as *complicated* hypertrophy; cases of *uncomplicated* hypertrophy being those in which the causative conditions are either not obvious or situated remotely from the heart.[1]

In complicated hypertrophy the antecedent and co-existing cardiac affections are those which involve over-repletion of the cavities, either in consequence of obstruction to the free passage of the blood through the orifices and vessels, or of regurgitation due to valvular insufficiency. The organ being unduly distended and stimulated by the accumulation of blood, its action becomes unduly forcible; the causes of accumulation being permanent and often progressively increasing, the increased action continues and augments, and hyper-nutrition is the result. The hypertrophy commences in that portion of the heart which is most directly affected by the complication, but the several portions sustain to each other, in their anatomical structure and functions, relations so close and reciprocal, that causes which at first are limited to one part, affect ultimately the whole organ. The enlargement, however, preponderates in the portion which is first affected. Directing attention with some detail to the mode in which lesions of the valves or orifices and vessels give rise to enlargement, we shall be led to consider the development of the affection in the different anatomical divisions of the heart, respectively, taking them up in the order of their greater relative liability to become hypertrophied. Of the several portions, the left ventricle, as already stated, is oftenest enlarged; next in liability to enlargement is the left auricle; next, the right ventricle, and, last, the right auricle.

[1] In 276 cases of enlargement in which hypertrophy predominated, Dr. T. R. Chambers (Decennium Pathologicum, *Brit. and For. Med.-Chir. Rev.*, vol. xii., 1853), found the heart free from valvular disease in 75, leaving 201 cases of complicated hypertrophy.

The lesions which especially lead to hypertrophy of the left ventricle, are those seated at the aortic orifice. Lesions in this situation may involve, as will be seen hereafter, contraction and consequently obstruction, or inadequateness of the valves and regurgitation of blood from the aorta into the ventricular cavity. Contraction and valvular insufficiency are not infrequently combined, causing, at the same time, obstruction and regurgitation. Either of these immediate effects of aortic lesions occasions over-repletion of the ventricle; hence, undue distension and stimulation, followed by undue force of the ventricular contractions, and, sooner or later, hyper-nutrition, usually accompanied with more or less dilatation. The enlargement due to the effects last mentioned, for a time is limited to the left ventricle. Eventually the other compartments become enlarged. The right ventricle is affected because each of the two ventricles participates in the action of the other. The two not only contract synchronously, but are in part composed of muscular fibres common to both. Hence, causes which either weaken or increase the force of the contractions of the one, exert, to a greater or less extent, a similar effect on the contractions of the other. Clinical observation shows that with enlargement of one ventricle, the other very rarely retains its normal size. This is a mode by which the enlargement is extended, applicable only to the ventricles. Another mode is more effective than this. The accumulation of blood within the cavity of the left ventricle offers an obstacle to the free transmission from the left auricle. The blood in passing from the auricle to the ventricle meets with an obstruction in the already repleted ventricle. Over-accumulation within the left auricle ensues; hence occurs, after a time, enlargement of the auricle. This enlargement involves generally more or less thickening of the walls, but dilatation here uniformly predominates over hypertrophy. Enlargement by hypertrophy, in fact, pertains exclusively to the ventricular portions of the heart. Persisting repletion of the left auricle offers an obstacle to the free transmission of blood from the lungs; hence arises congestion of the pulmonary vessels proportionate to the auricular accumulation, the latter being the greater, the more the auricular becomes dilated. Congestion of the pulmonary vessels offers an obstacle to the current propelled by the right ventricle into the pulmonary artery; hence, undue distension and excitement of the right ventricle, leading ultimately to enlargement of this portion of the heart. Over-accumulation and enlargement of the right ventricle offer an

obstacle to the passage of the blood from the right auricle into that cavity; hence result, at length, dilatation and thickening of the walls of the right auricle. Over-accumulation in this auricle induces congestion of the systemic and portal veins. This congestion offers an obstacle to the free passage of blood through the arteries of the larger circuit. Finally, this latter obstacle reacts on the left ventricle and adds to the accumulation in that compartment, where commenced the several links in the chain of sequences tending to the enlargement, successively, of all the other portions of the heart. And while the whole organ thus becomes implicated, the causes affecting primarily the left ventricle are more and more operative, giving preponderance to the enlargement of the latter. The enlargement of the left ventricle, and, sequentially, of the remainder of the organ, will be, *cæteris paribus*, proportionate to the duration and degree of the aortic contraction or insufficiency, or of both combined. Obstruction seated in the aorta either near or at some distance from the heart, such as is incident to aortic aneurism, leads to hypertrophy of the left ventricle primarily, and, subsequently, of the other portions. The effect is much more marked if the dilatation of the artery extend to the orifice rendering the valves inadequate, or if valvular lesions permitting regurgitation co-exist.

Enlargement commencing in the left auricle occurs in connection with lesions of frequent occurrence, affecting the mitral orifice and valves, and involving either contraction or insufficiency, or both these immediate effects. In auricular enlargements, however, as just stated, dilatation predominates over hypertrophy. Mitral obstruction and regurgitation lead to accumulation in the left auricle, the passage of the blood from the auricle to the ventricle being impeded by the one, and a reversed current from the ventricle to the auricle being incident to the other. Next follow pulmonary congestion and enlargement of the right ventricle, the same as when these results take place in cases of aortic obstruction and regurgitation. So far as the ventricles are concerned in connection with the mitral lesions mentioned, the right ventricle is first enlarged, and its enlargement often, if not generally, preponderates over that of the left ventricle, unless, as frequently occurs, aortic lesions also exist. The enlargement of the right, however, leads to that of the left ventricle, partly from the community in structure and in part from the ultimate effect on this ventricle of obstructive accumulation successively in the right auricle and systemic veins.

Contraction and valvular insufficiency at the pulmonary orifice occasion, primarily, enlargement of the right ventricle, precisely as aortic lesions induce, first, enlargement of the left ventricle. Lesions at the pulmonary orifice after birth, however, are so rarely met with that, practically, their occasional occurrence may almost be disregarded in diagnosis. In fœtal life, contraction at this orifice is not very infrequent. It is the point of departure for many of the congenital malformations of the heart. In these instances, the right ventricle is often found enormously hypertrophied.

Lesions at the tricuspid orifice being extremely infrequent, enlargement of the right auricle rarely occurs, except consecutively to an affection of the right ventricle. Over-accumulation in this ventricle involves obstruction and accumulation within the auricle with which it communicates, and the ulterior consequences already mentioned. The remote and incidental effects of obstruction to the circulation, except as regards the size of the heart, will be considered, in connection with the subject of valvular lesions, in another chapter.

Enlargement of the heart, uncomplicated with other cardiac affections, may be traced in some instances to obstruction at a distance from the centre of the circulation. Pulmonary obstruction, incident to emphysema of the lungs, and occasionally to chronic pleurisy, collapse, and dilated bronchi, leads to cardiac enlargement. In these cases, the point of departure is the right ventricle, and the enlargement of this portion preponderates over that of the other compartments.[1] Obstruction in the systemic vessels, occurring independently of prior disease of the heart, and sufficient in degree and persistence to give rise to enlargement of the heart, is not so easily determinable as pulmonary obstruction. It has been conjectured that in this way Bright's disease of the kidneys may lead to cardiac disease, these affections being not very unfrequently associated. It is, however, a question whether, in such instances, the affection of

[1] Dr. Gairdner, of Edinburgh, has suggested that enlargement of the heart, incident to emphysema and other affections of the lungs attended with diminution of their substance, may be produced by the dilatation of the chest in inspiration, and that obstruction of the pulmonary vessels plays a subordinate part in the enlargement. The suction force thus exerted of course cannot be made to explain hypertrophy, but only dilatation, nor can it be considered as acting on the right ventricle to the exclusion of other portions of the heart. Vide art. in *Brit. and For. Med.-Chir. Rev.*, July, 1853, entitled "Considerations on the causes of dilatation of the heart, with an analysis of evidence bearing on the connection of that affection with disease of the lung."

the kidneys be not consecutive to, and dependent upon, the affection of the heart. The changes which the arteries undergo in the latter part of life, by which their elasticity is impaired and their calibre diminished, are, with much reason, supposed to stand in a causative relation to enlargement of the heart in some cases. These changes, perhaps, in a measure at least, account for the progressively increasing size of the heart, which, according to the researches of Bizot, marks the progress from middle life to old age.

Cases of uncomplicated enlargement, as already stated, are few in comparison with the number of those in which the enlargement is associated with other and anterior cardiac lesions. If from the number of the former are excluded those referable to obstruction situated remotely from the heart, the residue is exceedingly small. Pushing still farther this elimination, and rejecting the cases in which hypertrophy is associated with dilatation, in other words, accepting only cases of simple hypertrophy, their occurrence is so rare that they may be classed among the curiosities of medical experience. The best specimen of simple, uncomplicated hypertrophy which has come under my observation, was obtained at the autopsy of a young unmarried female, who died after an abortion had been procured, in the latter part of pregnancy, by a practitioner of homœopathy, who was convicted of the crime and sent to the State prison. This female had apparently been well and vigorous until her pregnancy, when she became anasarcous. The kidneys presented evidence of granular degeneration. Death occurred just after delivery, during a convulsion. The heart in this case weighed fourteen and a half ounces; the thickness of the left ventricle was nearly an inch, and that of the right ventricle a fourth of an inch. None of the cavities appeared to be enlarged. Nothing was developed in the judicial investigation of the case to show that there had existed symptoms referable directly to the cardiac hypertrophy.

It was formerly supposed that prolonged functional disorder of the heart frequently eventuated in the development of hypertrophy. This opinion, sanctioned by Corvisart, is not sustained by clinical experience. It may be fairly doubted if the palpitation incident to anemia and other inorganic causes be ever competent, in itself, to induce hypertrophy. At first view, this statement may appear inconsistent with the fact that the abnormal growth of the muscular walls of the heart is the consequence of abnormal muscular action of the organ. This inconsistency disappears when it is considered

that functional palpitation, even when intense, does not involve that increase of power or strength of muscular action which is incident to the over-accumulation of blood from an impediment to the circulation. Moreover, the increased action from nervous excitation is never so constant and persisting as that due to valvular or other lesions which occasion obstruction. In the latter case, hypertrophy is the result of increased action, beginning imperceptibly and progressively increasing for many months, and even years.

In leaving this branch of the subject, it should be stated that, although in the immense majority of cases enlargement is referable to obvious lesions either within or without the heart, involving impediment to the circulation, a few instances are on record in which the organ attained to an enormous size, and no other lesions were discoverable. A case is cited by Jones and Sieveking in which the heart weighed five pounds, the valves being perfectly healthy, and no morbid appearances elsewhere discovered to account for the enlargement. Perhaps the most rational explanation which can be given of these cases is that, congenitally, the size of the heart is disproportionate to the capacity of the vascular system. This explanation was given by Laennec, and is adopted by Rokitansky.

The account which has been given of the manner in which the several compartments of the heart become enlarged is applicable, in a measure, to both forms of enlargement, viz., hypertrophy and dilatation. In the vast majority of the cases of enlargement by hypertrophy, it is to be borne in mind that the hypertrophy is accompanied by more or less dilatation. The causes which determine a predominance of dilatation will be more appropriately considered in a subsequent section of this chapter, devoted to the subject of "enlargement by dilatation." The same causes determine the degree of dilatation which accompanies hypertrophy when the latter predominates. In cases of enlargement by hypertrophy, the accompanying dilatation, according to the views of some writers, precedes the hypertrophy. It is more reasonable to suppose the reverse of this, *i. e.*, that the dilatation is consecutive to the hypertrophy. The first effect of over-distension and stimulation from an undue accumulation of blood is the increased growth of the muscular walls. In the healthy, vigorous action of the heart, the ventricles, probably in general, contract, so that the endocardial surfaces come into apposition, and the contents of the cavities are

completely expelled.[1] Over-repletion of the cavities excites a more forcible ventricular action, which for a time overcomes the obstruction inducing the repletion. Meanwhile, hyper-nutrition follows, and hypertrophy is produced. The increased muscular growth for a certain period protects against the occurrence of dilatation. At length, the hypertrophy reaches a limit when it increases slowly, if at all. The causes, however, persist, and perhaps become more and more operative. Dilatation then ensues, and from this period the progressive enlargement is due chiefly to augmentation of the cavities. This view is not only rational, but sustained by facts derived from clinical experience. Observation shows that, as a rule, in proportion to the duration of organic affections of the heart inducing enlargement, dilatation exceeds, relatively, hypertrophy; and, in the great majority of the cases in which death occurs, not from affections incidental to heart disease, but as a termination of the latter, dilatation predominates over hypertrophy. According to this view, hypertrophy becomes an important conservative provision, first, against over-accumulation of blood, and, second, against the more serious form of enlargement, viz., dilatation.

Hypertrophy with diminution of the size of the cavities claims a few words. Under the title of "concentric hypertrophy"[2] (first described and so named by Bertin), this was regarded formerly as a morbid condition occurring not very unfrequently. The investigations of Cruveilhier and others within the past few years have led some pathologists to reject it entirely as a morbid condition, and, it is generally conceded that, if it ever occurs, the instances are extremely rare. The ventricular cavities, in connection with increased thickness of the walls, are sometimes observed after death to be considerably diminished. This fact is not doubted; but it is supposed that both the diminished cavities and the thickened walls in such cases are due to an unusual degree of tonic contraction of the muscular fibres persisting after death. Cruveilhier found this appearance in the bodies of persons who had suffered death by

[1] That the inner surfaces of the ventricles come into contact, and with considerable force, was shown by an appearance presented in a heart contained in my collection. A rough, projecting, calcareous deposit existed on the anterior curtain of the mitral valve. Directly opposite, on the septum, over a space corresponding in size, as well as situation, to this deposit, the endocardium had become thickened and opaque, evidently due to the forcible pressure of the rough, calcareous mass. The ventricle was hypertrophied and dilated.

[2] Also called centripetal hypertrophy. (*Bouillaud.*)

decapitation. It has been observed in other cases after death from hemorrhage, and from diseases accompanied with much loss of fluids. In some instances, the contracted size of the cavities may be made to disappear by mechanical dilatation with the fingers, and it may disappear spontaneously some time after death, especially if the heart be macerated in water. The coexistence of contracted cavities and morbid thickness of the walls, is deemed inconsistent with the conditions giving rise to hypertrophy, and the mechanism of its production. The tendency of these conditions, in most cases, is, undoubtedly, to dilatation. Yet it is conceivable that causes which have induced hypertrophy without dilatation may cease, and that afterwards the tendency of the hypertrophy is to lessen the ventricular cavities. This is the more intelligible when it is considered that, according to the view which has been presented in the development of hypertrophy and dilatation, the former in point of time takes precedence. Hypertrophy of the left ventricle, with contraction of the cavity, may be accounted for in cases in which there exists either mitral contraction or regurgitation. This ventricle, under these circumstances, may become hypertrophied in the manner already considered, while, owing to contraction at the mitral orifice, or regurgitation, the accumulation within its cavity, instead of being sufficient to occasion distension, for a time, at least, is less than normal, and, therefore, the tendency of the hypertrophy, while this state of things continues, may be to contraction rather than dilatation.[1] Without discussing the subject, which does not possess much practical importance, the possibility of concentric hypertrophy must be admitted, while it is probable that, in the majority of the cases formerly so considered, the appearances after death do not fairly represent either the capacity of the cavities or the thickness of the walls during life. It is to be borne in mind that, in the cases in which unusual tonic contraction of the ventricles is suspected, the thickness of the walls may not be adequate evidence of the existence of hypertrophy. The weight of the heart is the test in such cases. If the weight exceed the limits of health, without reference to the size of the cavities or thickness of the walls, it is to be concluded that hypertrophy exists.

[1] This view is advocated by Professor M. Forget, of Strasbourg, *Précis Théorique et Pratique des Maladies du Cœur, etc.*, 1851, p. 247. Prof. F. contends that abnormal diminution is liable to occur whenever an obstruction exists, as regards the circulation, at a point behind (*en arrière*) the diminished cavity, the tendency to dilatation always existing if the obstruction be situated anteriorly (*en avant*).

Symptoms and Pathological Effects of Hypertrophy.

The symptoms of hypertrophy, in the cases which come under the cognizance of the physician, are generally intermingled with, and obscured by, those of the concomitant cardiac or other affections which have given rise to enlargement. Cases of simple, uncomplicated hypertrophy are so rare that its clinical history can hardly be said to have been established by observation. The symptomatic phenomena which are described as distinctive of it have been determined inferentially rather than by facts observed in well-authenticated cases. Rationally considered, it is clear that the symptoms would be those indicative of abnormal energy or power of the heart. Undue determination of blood to the head might be expected to occasion certain phenomena, such as cephalalgia, flushing of the face, throbbing, vertigo, etc. These symptoms have relation to hypertrophy affecting the left ventricle. Assuming the absence of aortic obstruction and of mitral regurgitation, the pulse would represent the power of the ventricular contractions by its force, fulness, and incompressibility. Dyspnœa, when, from any cause, the action of the heart is increased, as, for example, after exercise, would denote that the hypertrophy affected the right ventricle. Of the powerful action of the heart the patient would be conscious when his attention was directed to it, and it would be apparent from the movements of parts of the body and the dress. The digestive and assimilative functions would not be expected to offer any marked symptoms of disorder. The muscular strength would not be diminished, nutrition would not be impaired, nor the functions of secretion and excretion interrupted. This is a brief account of a hypothetical case of simple, uncomplicated hypertrophy. I am unable to give a description based on personal observation, or on an analysis of reported cases. The group of symptoms is not highly distinctive; the affection would be likely to be overlooked, and, if the hypertrophy were but moderate in degree, the immediate inconveniences would probably not be sufficient to lead the patient to seek for medical advice.

Associated with valvular lesions, emphysema, aneurism, and other antecedent and causative affections, the symptoms distinctly referable to hypertrophy are few. The cerebral symptoms are attributable to obstructed circulation rather than to an abnormal power of the heart. The same remark applies to dyspnœa and other

pulmonary symptoms. Valvular obstruction and regurgitation modify, in a marked degree, the characters of the pulse. In short, that which chiefly possesses significance is the evidence afforded by observation and the consciousness of the patient that the heart habitually acts with undue strength. To this the mind of the patient becomes accustomed, and he often appears unconscious of it, even when it is very marked on a physical examination of the præcordia. This evidence of hypertrophy lessens in proportion as it is accompanied by dilatation, and finally disappears when the latter predominates.

The pathological effects of hypertrophy are to be disconnected from those of concomitant affections and accompanying dilatation. Thus isolated, it is not easy to impute to it any special or very important pathological effects. It has been supposed that hypertrophy of the left ventricle sometimes leads to apoplexy and hemiplegia, due to extravasation of blood or congestion, in consequence of the force with which the current of blood is propelled into the vessels of the brain. That these cerebral affections occur as effects of disease of the heart is not to be denied, but the cardiac affections which more especially tend to produce them, are those involving obstruction to the return of blood from the head. Moreover, it is to be borne in mind that great hypertrophy of the left ventricle is generally complicated either with aortic obstruction or regurgitation, or both, and that, under these circumstances, the strain upon the coats of the cerebral arteries is not commensurate with the force of the ventricular contractions. Statistical researches show that the occurrence of apoplexy in connection with heart disease, is not proportionate to the degree of hypertrophy.[1] Hypertrophy of the right ventricle has also been supposed to give rise to hæmoptysis and pulmonary apoplexy. But clinical observation shows that these effects very rarely, if ever, take place, except when (as is often the case) with hypertrophy of the right ventricle, is conjoined contraction of the mitral orifice. The latter involves an impediment to the pulmonary circulation more likely to give rise to hemorrhage than the force with which the blood is propelled by the hypertrophied ventricle. Dropsical effusion into the areolar tissue and serous cavities (general dropsy) is a common effect of organic disease of the heart. It is not, however, an effect

[1] See Walshe on Diseases of the Lungs and Heart, second edition, for an analysis of cases collected from different authors, the results appearing to show that hypertrophy has little or no effect in determining the occurrence of apoplexy.

attributable to hypertrophy. Simple, uncomplicated hypertrophy would be incapable of producing it. When it occurs in connection with cardiac enlargement, it is due to obstruction from valvular disease or from dilatation.

Physical Signs and Diagnosis of Enlargement and Hypertrophy.

The physical signs of enlargement of the heart are common to both forms, viz., hypertrophy and dilatation. After having considered these signs in the present connection, it will only be necessary to refer to them briefly in treating of dilatation. Incidental to their consideration will be noticed the points distinctive of enlargement by hypertrophy. The different methods of physical exploration contribute evidence of cardiac enlargement. Enumerating them in the order of their relative importance, the methods available in the diagnosis are percussion, palpation, auscultation, inspection and mensuration. The signs obtained by these different methods may be conveniently classified and considered as follows: 1. Extended and increased dulness in the præcordia, as determined by percussion. 2. Altered situation and extent of the apex-beat; impulses elsewhere than over the apex of the heart; and abnormal force of impulse, as determined by palpation. 3. Abnormal modifications of the heart-sounds; diminished extent and degree of the respiratory murmur and vocal resonance within the præcordia, as determined by auscultation. 4. Enlargement of the præcordia and abnormal movements, as determined by inspection. 5. Increased size of the chest, as determined by mensuration.

1. *Extended and increased dulness in the præcordia as determined by percussion.*

It is obvious that the diagnostician must be acquainted with the extent and degree of the præcordial dulness due to the presence of the heart in health, before he is prepared to appreciate the signs of disease furnished by percussion. With reference to the results of percussion in health, the position of the heart and its anatomical relations to the lungs and the thoracic walls are to be considered.[1]

The heart is situated between the cartilages of the third and sixth ribs. The upper extremity, or base, is defined with sufficient

[1] Vide Fig. 1, Frontispiece.

precision by the upper margin of the third rib. The point or apex generally extends to the fifth intercostal space, near the junction of the rib to its cartilage. The organ is situated obliquely within the chest; a line passing through the longitudinal axis would intersect obliquely the clavicle near its acromial extremity. The median line and a vertical line passing through the nipple, are convenient landmarks for indicating the space which the heart occupies transversely. The median line divides the heart, leaving about one-third on the right and two-thirds on the left side. The left margin in the male extends to a point just within the nipple which is situated on the fourth rib near the junction of the rib with its cartilage. The point or apex is about three inches to the left of the median line, and an inch within a vertical line passing through the nipple. The right margin of the organ extends from half an inch to an inch beyond the sternum on the right side. Viewing the several portions of the heart in relation to the median line, on the right side are situated the right auricle and about a third of the right ventricle; on the left of this line are situated two-thirds of the right ventricle and the left auricle.

The relations of the heart to the adjacent organs are important with reference to the signs furnished by percussion and other methods of exploration. At the base are the large arteries connected with the ventricles, viz., the aorta and pulmonary artery, which extend upward beneath the sternum, the latter to the level of the upper margin of the second, and the former nearly as high as the first rib. The course of these vessels, and their respective positions relatively to each other, and to the thoracic walls, are of importance in regard to certain auscultatory signs, and will be referred to in that connection. The portion of the heart situated on the right of the median line is covered by the right lung.[1] The lower border of the organ, to the left of the median line, lies on the diaphragm, which separates it from the left lobe of the liver, and toward the apex from the stomach. Its relations to the stomach are more or less extensive, according to the degree of distension of the latter organ. The portion of the heart lying to the left of the median line is only partially covered by the left lung; a part is in contact (the pericardium of course intervening) with the thoracic walls. The space on the chest beneath which the heart is uncovered of lung, is called the *superficial cardiac region*. The space

[1] Vide Fig. 2, Frontispiece.

beyond this region occupied by the heart, situated beneath the right border of the left lung, is called the *deep cardiac region*. These names will often recur, and their import should be understood. The left lung extends downward on the median line to the level of the junction of the fourth costal cartilage with the sternum. From this point the border of the lung diverges, leaving an irregular quadrangular portion of the heart's surface exposed. This space may be embraced with sufficient precision for practical purposes within a right angled triangle, delineated as follows:[1] The oblique line, or hypothenuse, is drawn by connecting a point at the centre of the sternum on a level with the junction of the fourth costal cartilage, with the point where the apex of the heart comes in contact with the thoracic walls, usually in the fifth intercostal space, about an inch within a vertical line passing through the nipple, or about three inches to the left of the median line. The median line extending from the same point on the sternum, and a line extending transversely from the point of apex-beat to meet the median line, will form the two other sides of the triangle. The superficial cardiac region is thus bounded on two of its sides by lung, and on the greater part of one side, viz., the lower, by the liver and stomach, with the diaphragm intervening. The limits to which the deep cardiac region extends beyond those of the superficial cardiac region, have been already defined in giving the boundaries of the space which the heart occupies within the chest.

This account of the situation and anatomical relations of the heart, based on examinations of the dead subject, is sufficiently exact for practical purposes; but in the living body, it is to be borne in mind, the position of the organ relatively to the thoracic parietes and the adjacent organs varies within certain limitations, not only in different persons, but in the same person at different times. The size of the organ is variable, owing to a greater or less accumulation of blood in its cavities, more especially in the auricles. The whole organ is movable to some extent. The base is comparatively fixed, but the apex moves freely in a lateral direction, and varies its position in different postures of the body. The superficial cardiac region is larger or smaller according to differences in different persons as regards the volume of the left lung and the conformation of the chest. It is small in robust persons with deep chests, and larger in the slender and broad-

[1] Vide Fig. 1.

chested. Its size is greater at the close of an expiration than after an inspiration, and the difference is, of course, marked in proportion as these respiratory acts are forced. These are variations irrespective of those occasioned by disease. Moreover, in the dead subject, the conditions of the heart and lungs affecting their mutual relations are by no means uniform. The lungs collapse and shrink away from the heart more or less, according to contingencies which are independent of disease, and the state of the heart, as regards the quantity of blood remaining in its cavities, depends on the mode of dying and other circumstances. But happily these variations are not sufficient to render unreliable the signs incident to diseases of the heart.

During life, the space within which the heart in health is uncovered of lung and in contact with the chest, in other words, the limits of the "superficial cardiac region," and the boundaries of the heart beyond these limits, or the "deep cardiac region," may be determined by means of percussion. With sufficient care and practice, the two regions just named, to the left of the median line, may be determined on the chest in the majority of persons. Their limits, in fact, are often so distinctly definable that, in view of the changes which occur in the heart and lungs after death, the dimensions obtained by percussion during life represent more fairly the normal relations of these organs than measurements with the parts exposed to view in the dead subject. The limits of the superficial cardiac region are best ascertained by light percussion, commencing at the centre of the region. The upper limit in seventeen healthy persons in whom it was carefully ascertained was the cartilage of the fourth rib; in some the upper and in others the lower margin of the cartilage near the sternum. The outer limit on a transverse line passing through the nipple is at a point varying from half an inch to an inch and a half within the nipple, the average distance in twenty-two persons being a small fraction ($\frac{1}{8}$th) over an inch. The apex-beat, which is generally either seen or felt, determines the outer limit at the base of the triangle. The percussion-sound at this point is sometimes tympanitic from transmitted gastric resonance, the quality and pitch of sound denoting its source. In determining the lower boundary of the region, the line of demarcation between the liver and the lower border of the heart is to be distinguished by the percussion-sound. This, which Dr. Walshe calls "one of the most difficult practical problems in the art of percussion," is readily done in most persons. Percussing from a

point over the liver towards the heart, viz., in the epigastrium in a direction upwards and outwards to the left, the flat, short, high, liver-sound, at a little distance above the xiphoid cartilage, gives place to a sound dull but not flat, longer and lower in pitch. Connecting now the several points, already marked on the chest with ink or some coloring substance, we have a diagram representing the superficial cardiac region sufficiently exact for ascertaining its normal dimensions in the living subject. The average transverse diameter, measured from the median line to the outer limit, a little below the level of the nipple, in twenty-three healthy persons, was a small fraction over three inches, the maximum width being four, and the smallest two and a half inches. The average vertical diameter, measured on the median line in sixteen healthy persons, was two and a half inches, the maximum three, and the minimum one and three-quarter inches.

In determining the boundaries of the heart beyond the limits of the superficial cardiac region, that is, the extent of the "deep cardiac region," or, in other words still, the border of the præcordia, forcible percussion is requisite, but not force enough to occasion pain. In mapping on the chest this space, the course enjoined by Bouillaud has decided advantages, viz., commencing at some distance from the heart and percussing towards the præcordia. The points at which the percussion-sound is modified, *i. e.*, distinctly dull, being marked and connected by lines, the space occupied by the heart is delineated on the chest; and if the limits of the superficial cardiac region are delineated on the same chest, we have two concentric diagrams representing the two regions. Attention to the pitch of the percussion-sound is of great assistance in appreciating the dulness within the deep cardiac region, a change in this respect being more readily recognized than the difference in the degree of resonance. Taking the nipple as a landmark, in twenty-five healthy persons (all males) the left border was precisely at the nipple in sixteen; in six instances, it was within the nipple, the greatest distance being seven-eighths of an inch, and the smallest three-eighths; in three instances, it was without the nipple, being half an inch beyond in two, and three-eighths of an inch in the remaining instance.[1] The præcordial region, as determined by percussion on the living body, in the majority of instances, extends

[1] It should be stated that these, as well as the preceding results of percussion, were obtained by percussing while the persons were in a sitting posture.

somewhat farther to the left of the sternum than when this region is viewed in the dead subject, a fact doubtless owing to the presence of a larger quantity of blood within the cavities of the left side of the heart during life. On the right side of the sternum, on a level with the nipple, dulness is generally appreciable within a space varying from half an inch to an inch. The percussion-sound over the third rib near the sternum is generally sufficiently modified on percussing from above downwards to indicate the base of the heart in this situation.[1]

The foregoing details, which have been given as succinctly as possible, are essential as constituting the basis of the physical signs of enlargement of the heart. The latter, after these preliminaries, may be briefly presented. The area of præcordial dulness exceeds the limits of health in proportion as the volume of the heart is abnormally increased. The effect of an enlarged heart is especially manifest in the superficial cardiac region. The heart, in proportion to its augmented bulk, pushes aside the borders of the lungs, leaving a larger portion of its anterior surface uncovered and in contact with the thoracic walls. The superficial cardiac region becomes, of course, proportionately larger than in health. This effect is certainly the rule, and the exceptional instances described by some writers[2] in which the heart buries itself beneath the lungs, leaving its anterior surface covered to the same extent as in health, must be extremely rare, assuming the volume of the lungs to be normal. The enlargement of the superficial cardiac region is especially marked transversely to the left of the median line, owing to the heart increasing more in width than in length. The apex of the organ is generally removed to the left of its normal situation,

[1] The combination of percussion and auscultation, or auscultatory percussion, as proposed and practised by Drs. Cammann and Clark, of New York, is undoubtedly well adapted to determine with ease and accuracy the boundaries of the heart. See *New York Journal of Medicine*, July, 1840. That this mode is not more generally employed is because percussion, as usually practised, suffices for ordinary practical purposes. The stethoscope recently invented by Dr. Cammann is well suited to auscultatory percussion. The publication by Drs. Cammann and Clark just referred to, contains the average dimensions of the space occupied by the adult heart in a series of examinations. The following are the mean results :—

	Male.	Female.
Vertical diameter . .	4 in. 0 lines	3 in. 7 lines
Transverse " . .	4 " 4 "	4 " 1 line
Right oblique " . .	4 " 10 "	4 " 10 lines
Left oblique " . .	3 " 10 "	3 " 7 "

[2] Traité de Diagnostic, par Racle.

owing partly to the oblique position of the heart, and in part to the fixedness of the base of the organ, the latter, with the diaphragm below, offering mechanical resistance to much extension in a vertical direction. The apex being free, is moved readily in a lateral direction. The evidence, therefore, of the heart being abnormally uncovered of lung, and of the extent of its surface in contact with the chest, is obtained by percussing from the median line towards the nipple and towards the point where the apex-beat is felt. The lateral diameter of the superficial cardiac region at the inferior boundary, *i. e.*, between the median line and point of apex-beat, may be one, two, and even three inches greater than in health. The superficial dulness instead of ending an inch within a vertical line passing through the nipple, extends to this line, or from one to two inches beyond it. The presence of the apex-beat enables us to determine the diameter in this situation without practising percussion. This point may be more or less lowered, as well as carried to the left. It is frequently found in the sixth, and sometimes even in the seventh intercostal space, the inferior boundary of the superficial cardiac region being, of course, proportionately lower than in health. Percussing next from the left margin of the sternum on or just below the level of the nipple, the superficial dulness may be found to extend to the nipple, or half an inch, an inch, or even farther, beyond it. The diameter of the region here will correspond to the abnormal width of the heart. Other things being equal, the enlargement of the heart transversely may be accurately measured by the extent to which the diameter of the superficial cardiac region in this situation is increased. But it is to be borne in mind that the normal situation of the outer limit of this region is not the same in all persons. The average distance within the nipple is very nearly an inch, but the variation within the range of health, as has been seen, is from half an inch to an inch and a half. If the superficial dulness extend to within half an inch of the nipple, or possibly even within a still shorter distance, it may not be due to abnormal enlargement; and, on the other hand, in a person whose heart is normally covered by lung an inch and a half within the nipple, superficial dulness extending to a point within half an inch of the nipple would denote considerable enlargement of the heart. If the area of superficial dulness proper to the individual be not known, an abnormal increase of its dimensions cannot in any case be assumed unless the lateral diameter extend nearly or quite to the nipple. Here, as in other

instances, the extreme limits of healthy variation are of greater practical consequence than averages. In determining, however, whether the heart be enlarged or not, the distance from the apex to the median line is to be taken into account, and also the signs obtained by other methods of exploration than percussion.

The degree of dulness within the superficial cardiac region is, in general, greater than in health in proportion to the enlargement. In health, a portion of the heart is imbedded in lung sufficient to occasion the transmission of more or less pulmonary resonance over the whole of the præcordia. The degree of normal dulness differs in different persons. It is generally marked, and sometimes approaches to flatness. It is sufficient to render the limits of the region distinctly definable, except when great obesity exists, or, in the female, when the mammary development is unusually large. It is sufficiently intelligible that, in proportion as the lung is pushed aside in cases of enlargement, the dulness will be greater in degree than in health. In some instances it amounts to flatness. It is equally obvious that the sense of resistance felt in practising percussion will be marked according to the increased bulk of the heart.

It is important to bear in mind that increased extent and degree of superficial dulness are signs of enlargement of the heart, with this provision, viz., that the lungs are free from disease. The size of the area is affected by abnormal conditions of the latter organs, as well as the heart. In phthisis, the left lung is frequently contracted, so that the anterior margin is removed towards the border of the heart, leaving a larger portion of the heart's surface in contact with the thoracic walls, even though the size of the organ may be less than in health. A similar result follows chronic pleurisy, the lung not expanding, and resuming its normal volume sufficiently to cover the heart as in health. Happily, in these exceptional cases the liability to error is slight, for the existence of tuberculosis is determined without difficulty, and the retrospective diagnosis of pleurisy is also easily made. The præcordial space is not enlarged, and all doubt is removed by defining the boundaries of the deep cardiac region.

The relations of the heart and lungs are also affected by a variety of causes, irrespective of morbid conditions of either of these organs, such as enlargement of the liver, dilatation of the stomach, aneurism of the aorta, enlarged spleen, ascites, pregnancy, tumors in the mediastinum, etc. These disturbing causes are generally determinable;

and the importance of not limiting exploration to the præcordia, but extending the examination over the chest and abdomen in order to exclude these and other affections which alter the normal disposition of the heart and lungs, is sufficiently obvious. Errors of diagnosis are sometimes attributable to neglect of this precaution.

The limits of deep dulness are not extended beyond those of superficial dulness proportionately to the degree of enlargement of the heart, but it is sometimes desirable to ascertain the actual space which the heart occupies. Percussing from without the heart toward the præcordia, the lateral borders of the organ may generally be determined without great difficulty, and delineated on the chest. The enlargement of the deep cardiac region is not only manifested by dulness extending more or less without the left nipple, but also beyond its normal boundary to the right of the sternum. Not only the extent of this region, but the form of the heart may be delineated, and the latter is of diagnostic significance as respects the discrimination between hypertrophy and dilatation, the latter increasing more than the former the width in proportion to the length of the heart.

The evidence afforded by percussion of enlargement of the heart is much less marked, if, in conjunction, the left lung be affected with emphysema. This combination is not infrequent. The effect of emphysema of the left lung is to lessen and even abolish the superficial cardiac region. The anterior border of the lung may be extended forward so that the whole surface of the heart is covered. The heart, too, is often depressed below its normal situation by the pressure of the dilated lung. The co-existence of emphysema, thus, renders the area of the superficial cardiac region no longer an index of the existence and the degree of enlargement of the heart. The limits of the deep cardiac region are alone to be depended on, and they are not always, under these circumstances, easily defined. The combination renders the diagnosis difficult by impairing also concurrent signs of enlargement obtained by auscultation, inspection and palpation. Moreover, the symptoms of emphysema are liable to be confounded with those which are due to disease of heart. The individual cases in which this difficulty in diagnosis exists are easily recognized, for the signs of emphysema are sufficiently explicit; and in a certain proportion of these cases the diagnostician must be content to rely in a great measure on the well-known pathological association of the two affections, determining the relative proportion of each approximatively.

Enlargement of the heart results from different pathological conditions. In addition to the two forms, to the consideration of which this chapter is devoted, viz., hypertrophy and dilatation, the organ acquires an abnormal size from the accumulation of blood within its cavities and the deposit of morbid products and fat on its surface. The question may be here raised, whether percussion furnishes data for the differential diagnosis of the different varieties of enlargement. Hypertrophy or dilatation, as has been seen, may be limited to portions of the heart, or may disproportionably affect certain portions. It is stated that the dulness extends more to the left of the median line when the left ventricle is the seat of enlargement, and is more manifest on the opposite side when the right ventricle is affected. The relations of the two ventricles, however, is such that, in view of the position of the heart and the movableness of the body and apex, the left border is extended in proportion as the right side is increased in size; and it may fairly be doubted whether, as a rule, the foregoing statement holds good clinically. The right or left auricle, belonging to the base which is comparatively fixed, when considerably enlarged, may occasion a greater relative extent of dulness on the corresponding side of the sternum. This remark is also applicable to distension of the cavities of the heart by the accumulation of blood. Great distension of the right side of the heart, which occurs in some cases of obstruction to the pulmonary circulation, may be manifested by an abnormal extent of dulness over the site of the right auricle; and this extent of dulness may be found to have diminished when the causes of obstruction are removed. The ability to distinguish between hypertrophy and dilatation by the percussion-sound is more than questionable. This is a nicety which the student should not attempt to acquire, for in proportion as he might imagine that he had made the acquisition, would be his liability to error in practically trusting to it. The same remark is applicable to the endeavor to determine by percussion that enlargement of the heart is due to the deposit of fat or morbid products on its surface. Very considerable enlargement in a transverse direction of the superficial and deep cardiac regions, however, is presumptive evidence that the increased bulk is due to dilatation rather than hypertrophy, for the former, more than the latter, tends to increase the width of the organ and also to give rise to excessive augmentation of size. On the other hand, if percussion show that the heart is considerably lengthened, and that the transverse enlargement is not to much extent disproportionate to the

vertical, the presumption is in favor of hypertrophy rather than dilatation.

Enlarged extent and degree of præcordial dulness are produced by liquid accumulation within the pericardial sac, as well as by enlargement of the heart. Both may co-exist, and then the evidence afforded by percussion of cardiac enlargement ceases to be available. The points of distinction between the præcordial dulness due to liquid accumulation within the pericardial sac, and that due to enlargement of the heart, are important, and will be considered in connection with the subject of pericarditis.

2. *Altered situation and extent of the apex-beat; impulses elsewhere than over the apex of the heart, and abnormal force of impulse, as determined by palpation.*

The point at which the apex, or pointed extremity of the heart, presses with an impulsive force against the thoracic walls, is in the fifth intercostal space, the person examined being in the sitting posture. Of twenty-five healthy persons examined, none presented an exception to this rule. In this intercostal space, the impulse is felt over an area varying from half an inch to an inch and a half, in health. The average transverse diameter of this area, in thirteen persons, was a fraction ($\frac{1}{14}$th) over an inch. The centre of this area, where the force of the beat is greatest, is situated within a vertical line passing through the nipple, at a distance from that line varying from two inches to three-eighths of an inch, the average, in eighteen persons, being a fraction ($\frac{1}{5}$th) over an inch. The distance from the median line to the centre of this area varies between three inches and five-eighths and two inches and five-eighths, the average, in fifteen persons, being a fraction ($\frac{1}{13}$th) under three inches. Measured from a transverse line passing through the nipple, the distance varies from an inch and an eighth to two inches, the average, in eight persons, being a fraction ($\frac{1}{7}$th) over one and a half inch. These are the relations of the apex-beat in the sitting posture. Deviations take place when the posture is changed, owing to the movableness of the apex and body of the heart. In the recumbent position on the back, the beat is frequently felt in the fourth intercostal space, the same relations laterally to the nipple and median line as in the sitting posture being preserved. The frequency with which this is observed has led some late writers to state, incorrectly, that, as a rule, the apex-beat is in the

fourth intercostal space.[1] Lying on the right side, the centre of the area within which the beat is felt is removed about half an inch nearer the sternum. Lying on the left side, the beat is removed to the left, so that the centre of the area generally falls on a vertical line passing through the nipple, and the impulse is felt half an inch without this line. The respiratory movements sometimes affect the situation of the apex-beat. I have not observed it to be lowered by a full inspiration, but it is occasionally raised from the fifth to the fourth intercostal space by a forced expiration, the persons examined in the sitting posture. The apex-beat is not unfrequently inappreciable to the touch in healthy persons, in the sitting posture. The persons in whom it is wanting have generally deep chests. Thickness of the soft parts also prevents it from being felt. It is lost in the recumbent position on the back in some instances in which it is felt when the person is sitting. It is still oftener lost when the person lies on the right side, but very rarely when the position is on the left side. In the latter position it is sometimes felt when not appreciable in any other.

The force of the impulse varies in different persons. It is rarely strong when the person is tranquil and free from mental agitation. It is generally quite feeble. It is almost invariably less when the person lies on the back than in the sitting posture; and it is still more diminished, when not lost, if the position be on the right side. Lying on the left side increases the impulsive force; the beat is strongest in this position. The sensation on applying the fingers over the area of the apex-beat, as remarked by Dr. Walshe, is that of a gliding as well as an impulsive movement. It is not that of a percussion or blow. It is sufficiently clear, on a little reflection, that the apex of the heart does not withdraw itself from the thoracic walls, and then come into forcible contact through an open space. The pressure of the atmosphere on the exterior surface is sufficient to prevent the heart receding from the chest, except so far as it is displaced by intervening pulmonary tissue. The beat must, of necessity, be produced by movements incident to the changes in form of the organ, and not to the tilting forward of the apex, as was formerly imagined.

The mechanism of the heart's impulse has been a fruitful theme for discussion. It does not fall within the scope of this work to consider the various theories which have been proposed. The

[1] Verneuil, 1852, Racle, op. cit.

subject, however, naturally presents itself in this connection, and claims a few remarks. It is generally admitted that the beat occurs synchronously with the systolic contraction of the ventricles. This is denied by some, and a theory which attributes it to the shock of the current of blood propelled into the ventricles by the contraction of the auricles, numbers among its supporters several distinguished names. Without discussing this theory, the improbability that the auricles possess sufficient power of contraction to account for the phenomena pertaining to the impulse, and the fact that the beat and the pulsation of the large arteries near the heart (*e. g.*, the carotids) occur without any appreciable interval of time, together with the observations of vivisectors, seem to render it conclusive that the commonly received doctrine is correct.[1] It may also be assumed that the impulse is produced by the apex of the heart, as, in fact, is assumed in the expression "apex-beat." The question, then, resolves itself into this: In what manner do the systolic movements of the heart cause the apex to press with a certain degree of impulsive force against the thoracic walls? This question is in a great measure answered if it be conceded that the apex of the heart is elongated during the ventricular systole. Some of the older anatomists, Galen, Vesalius, Harvey, and, later, John Hunter, entertained this view; while it was denied by Steno, Lancisci, Haller, and others. More recently, Dr. Hope and others, resorting to vivisections practised on animals of large size, were led to conclude that during the systolic contraction of the ventricles the heart is shortened by an approximation of the apex towards the base. It is difficult to understand how careful observers should be deceived in this regard, but, in the mind of the author, this conclusion is undoubtedly erroneous. Drs. Pennock and Moore, in their vivisections in 1839, satisfied themselves that the heart elongates during the ventricular contractions, and they even measured the extent of elongation. For several years, Prof. Dalton, of New York, has been accustomed, in his courses of instruction in Physiology, to demonstrate this fact, and the author has had an opportunity of witnessing a demonstration by him on an animal of considerable

[1] The reader, desirous of knowing the grounds on which the diastolic theory of the heart's impulse is sustained, is referred to the treatise on *General Pathology*, by Prof. Alfred Stillé; and for a still more elaborate exposition to the *Traité Experimental et Clinique d'Auscultation*, par J. H. S. Beau, Paris, 1856. A review of the latter by the author is contained in the *American Journal of Medical Sciences*, No. for January, 1857.

size (a sheep), while engaged in writing this chapter. The systolic elongation of the heart is therefore assumed in this work, in opposition to the statements of most, if not all previous writers on the diseases of this organ.[1] In elongating, the heart performs a revolving or spiral movement from left to right.[2] It is thus easy to perceive that the extremity of the organ presses against the chest with an impulsive, boring movement, more or less forcible, according to the power of the ventricular systole. Admitting the correctness of these statements, the mechanism of the impulse seems sufficiently explained; but the inquiry arises, is the elongation of the apex due directly to the muscular contraction of the ventricles, or to an intermediate force derived from the blood impelled by the systole against this extremity of the heart? A German theory, ascribed to Gutbrod, but, according to Dr. Markham, proposed by Dr. Alderson in an English quarterly journal as long ago as 1825, attributes the impulse to a reversed current of blood within the ventricles. This is known as the "Segner's water wheel," and the "recoil" theory. It is adopted by Prof. Skoda, and by the author of a late English work on diseases of the heart, Dr. Markham. The explanation of the impulse, according to this theory, is thus given by Dr. Gutbrod:[3] "It is a well-known physical law, that when a fluid escapes from a vessel, the equality of pressure produced by the fluid on the walls of the vessel is lost, for there is no pressure at the opening whence the fluid escapes; but at that part of the vessel which is opposite to the opening, the pressure is still exerted. This pressure it is which sets Segner's wheel in motion, produces the recoil of firearms, etc. By contraction of the ventricles, the pressure which the blood exerts upon the walls of the heart, opposite to the opening whence the stream escapes, causes a movement of the heart in a direction contrary to that of the stream of blood, and by this movement the impulse of the heart against the walls of the thorax is produced. The heart is driven in a direction contrary to that of the arteries, with a force proportionate

[1] The modern revival of the belief in the systolic elongation of the heart, may be characterized as the American doctrine. It does not appear to have been as yet adopted on the other side of the Atlantic. Vide *Dalton's Physiology*, Philadelphia, 1859.

[2] The movement of the apex from left to right with the ventricular systole, is sometimes very apparent when the thoracic walls are much thinned by emaciation. Vide case of M., Private Records, vol. x. page 648.

[3] A Treatise on Auscultation and Percussion, by Dr. Joseph Skoda. Translated by Dr. Markham.

to the quantity and the velocity of the current of blood." This theory is controverted by different writers;[1] but without entering into a discussion of its merits, it is rendered gratuitous by the fact that the elongation of the heart occurs when its cavities are entirely free from blood. If the heart be quickly removed from a living animal, the auricles opened and the organ placed in a vertical position with the base downward, the contractions of the ventricle continue for several minutes, and the elongation in this experiment is conspicuously manifest.[2] This shows conclusively that the elongation takes place independently of the current of blood.[3]

Directing attention now to the signs of enlargement and of hypertrophy obtained by palpation, those relating to the situation of the apex-beat are to be first noticed. The apex-beat is carried to the left of its normal situation and frequently lowered when the bulk of the heart is increased. These changes are among the most constant and reliable of the signs of enlargement. The beat may be felt one, two, or even three inches without the nipple. It may be found in the sixth and even in the seventh intercostal space. The distance to which it is removed in these directions, assuming that the alterations depend exclusively on the increased bulk of the heart, constitutes a criterion for estimating the amount of enlargement. It must, however, be considered that abnormal conditions extrinsic to the heart alter the relations of the apex to the walls of the chest, such as enlargement of the left lobe of the liver, distension of the stomach, ascites, enlarged spleen. These and others

[1] Vide note to French translation of Skoda's Treatise, by the translator, Dr. Aran.

[2] For an illustration of this experiment, vide Dalton's Physiology.

[3] Prof. H. Bamberger, of Wurzburg, has reported a case in which a healthy man attempted to commit suicide by stabbing himself in the breast with a sharp knife. The wound was at the lower margin of the fifth rib, within the nipple, and the man had evidently selected the spot where the heart's impulse was felt. On pressing his finger into the wound, Prof. B. felt the apex of the heart distinctly, the pericardium evidently having been opened. He availed himself of the opportunity to study the movements of the heart, and he states as follows: "When my finger was introduced from the point toward the back, I could convince myself with the greatest certainty that at every systole the hardened and pointed apex slipped down along the front wall of the chest, somewhat to the left and a little below the lower margin of the wound, whilst in the diastolic movement the apex retreated upward and could not be felt." This observation, which affords valuable confirmatory evidence of the systolic elongation of the heart, is not offered by the reporter as such; he regarding it as settled that the vertical diameter of the organ is shortened in the systole.

pertaining to the abdomen may remove the apex to the left, but without lowering it. An aneurismal or other tumor situated above the heart may give rise to the same change with depression. An emphysematous left lung pushes the heart downwards, but generally towards the epigastrium, often giving rise to an impulse in this situation, while the normal apex-beat is suppressed. These extrinsic conditions are, of course, to be excluded before the abnormal position of the apex can be regarded as a sign of enlargement of the heart. The limits of variation in health, in different positions of the body, are to be borne in mind in deciding whether the situation be normal or abnormal. If the patient be examined in the sitting posture, and the apex-beat be found in the fifth intercostal space, it is not lower than natural; but if the patient lie on the back, the chances are about equal that if the beat be in that space it is lowered; but if it be abnormally lowered, it will also be removed to the left in the great majority of cases. With reference to its relations laterally, it may be within half an inch of a vertical line passing through the nipple, or three and a half inches from the median line, without exceeding the range of healthy variation. If on a line with the nipple, or four inches from the median line, its situation is abnormal, provided the patient be either sitting or lying on the back. In the majority of the cases of enlargement which come under the notice of the physician, it is found without the line of the nipple.

The area in which the apex-beat is felt (averaging about an inch in health) is extended in cases of enlargement of the heart. The extremity of the organ is less pointed than in health; it is blunt or rounded, and consequently a broader surface comes into contact with the thoracic walls during the systolic impulse. This is a sign of some importance taken in connection with other signs denoting enlargement either by hypertrophy or dilatation.

In cases of hypertrophy of the left ventricle, the force of the apex-beat is abnormally great in proportion to the increased thickness of the walls, provided that the form of the apex be not greatly altered, the muscular power of the organ not weakened, or the completeness of the ventricular contractions not prevented by contraction at the aortic orifice, or other causes. An abnormal force of the apex-beat is associated with change in situation and extension of the area in which the beat is felt. The force of the beat thus associated is an important sign as showing that the enlargement is due to hypertrophy rather than to dilatation, or that the

former predominates. In proportion as the left ventricle is hypertrophied rather than dilated, other things being equal, the force of the beat is augmented. Augmented force of the beat, however, may be due simply to increased muscular activity of the organ without enlargement. The heart is affected functionally or dynamically without organic disease. The beat is augmented in the same manner as under excitement by active exercise or mental agitation. How is it to be determined whether the abnormal force of the beat be due to hypertrophy or simply to morbid excitement of the organ? The sensation in the latter case is that of increased *action*, and in the former case of increased *power*, of the impulsive movement. This distinction is generally appreciable. The beat in hypertrophy is felt to be produced by a powerful contraction of the ventricle; the impression conveyed by the touch is that of a prolonged, sluggish, as well as strong impulse. In mere functional excitation, the beat is more abrupt, quick, and brief, giving the idea of violence rather than of strength. The distinction is important, and would be vastly more so were the discrimination to rest solely on the difference, as respects the force of the beat. But in hypertrophy there are the coexisting signs of enlargement which are wanting in an affection simply functional. Increased force of the apex-beat is by no means a constant sign of hypertrophy. On the contrary, the beat may be suppressed. This may depend in part on the change of form which the extremity of the organ undergoes, and partly on the weakness incident sometimes to enlargement, even when the muscular tissue is augmented. The latter belongs to a late period in the progress of the disease. Suppression of the apex-beat is much more apt to occur in cases of dilatation than in cases of hypertrophy, because in the former the extremity of the organ is more blunted and weakness more marked.

Other signs of cardiac enlargement and hypertrophy than those relating to the apex-beat are obtained by palpation. Abnormal impulses may be felt in other situations than over the apex. Occasionally, in health, in addition to the apex-beat in the normal situation, an impulse is appreciable in the fourth intercostal space, and, in some instances, in the epigastrium to the left of the xiphoid cartilage. A double impulse, viz., in the fourth and fifth, or in the fifth and sixth intercostal spaces, is not unusual in cases of enlargement, and especially enlargement by hypertrophy. In some cases impulses are felt in three and even in four intercostal spaces. In these cases the lowest point of impulse is the farthest removed from

the median line, and the impulses above are, severally, situated nearer the sternum. In a patient under observation at the time of writing, impulses are felt and seen in the fifth, fourth, third, and second intercostal spaces. The impulse in the fifth intercostal space is situated an inch without a vertical line passing through the nipple; that in the fourth, is just within the nipple, and the impulses in the third and second spaces are near the left margin of the sternum.[1] The explanation of these additional impulses in cases of enlargement is, the heart being in contact with the thoracic walls over a larger space than in health, in other words, the area of the superficial cardiac region being enlarged, the movements of the organ are communicated to the yielding spaces between the ribs. This does not take place, as a rule, in health, in consequence of the interposition of lung save over a comparatively limited space. The impulsive movements, elsewhere than over the apex, are not always coincident with the ventricular systole; in other words, the elevations or outward motions at the several points at which the movements are observed, do not take place in unison, but in some instances in alternation. Thus when movements are felt in the fourth and sixth intercostal spaces, that in the sixth is the apex-beat and systolic, while that in the fourth may occur with the diastole of the ventricles. Alternation of the impulsive movements in these two intercostal spaces, is not unfrequently observed in cases of hypertrophy. The superior or diastolic movement was called by Dr. Hope the back-stroke of the heart. It is stated that this is sometimes observed in healthy persons when the heart acts with unusual vigor.[2] Generally in the cases in which a diastolic movement is observed, retraction of the intercostal space takes place during the ventricular systole, due to the flattening of a portion of the heart, and the movement of impulsion which alternates with the apex-beat is, in fact, only the elevation of the space to the level from which it was depressed. In other words, the space over the body of the heart yields to atmospherical pressure and follows the retreating ventricular walls during the systole, resuming its level when the heart assumes a more globular form during the diastole. The impulsion is not strong, and may be visible when not distinctly felt. In the case just referred to in which four distinct points of impulse are observable, the impulsion

[1] Case of Bergmann. Hospital Records, vol. xiii.

[2] Bellingham on Diseases of the Heart. Dublin, 1853. Part I. p. 81.

in the fifth, third and second intercostal spaces appears to take place during the systole, and that in the fourth intercostal space during the diastole of the ventricles. The three former are stronger than the latter. An impulse over, or a little below the base of the heart, *i. e.*, in the third and possibly in the second intercostal space, is referable to the expansion of the upper portion of the organ during the systole. The fact of this expansion and the force with which it takes place are shown by grasping the heart near the base in a living animal. A strong pressure is felt when the ventricles contract. It is not difficult to understand that the change of form at the base should communicate an impulsive movement to the intercostal space when the heart is abnormally uncovered of lung, and also in some instances of palpitation without organic disease, when the action of the heart is notably augmented. It is possible that, in some instances, the dilatation of the pulmonary artery following the systole of the ventricles, or the shock produced by the sudden arrest of the column of blood in consequence of the expansion of the sigmoid valves during the ventricular diastole, may give rise to an impulsive movement which may be felt in the second left intercostal space. Dr. Sibson states that a diastolic impulse is sometimes felt in this situation when, from pulmonary disease, the left lung recedes at this point, leaving the artery uncovered and in contact with the parietes of the chest.[1] An impulse situated here, referable to the pulmonary artery, is more likely to occur, for obvious reasons, in cases of hypertrophy of the right ventricle and when there exists obstruction to the pulmonary circulation. Laennec entertained the idea that an impulse on the left side at the base of the heart was sometimes due to the contraction of the left auricle. Aside from the fact that the greater part of the auricle is covered by the large arteries emerging from the heart, and the improbability of its ever contracting with sufficient force to communicate a perceptible impulse to the walls of the chest, it is difficult to understand how any other than a movement of retraction can accompany its systole. It seems far more reasonable to attribute an impulse in this situation, either to the ventricles or to the pulmonary artery. If there be free regurgitation through the mitral orifice, it is intelligible that the retrograde current of blood impelled by the force of the systole of the left ventricle may occasion an impulse over the auricle. This is perhaps the explanation

[1] Medical Anatomy.

in some instances, at least, in which an auricular impulse has been supposed to exist. Dr. Stokes reports a case in which an impulse was felt on the right side of the sternum, evidently, from the appearances after death, due to a retro-current through the tricuspid orifice, the right auricle being enormously dilated and its walls attenuated.[1] It is evident that an impulse produced in this way through the left auricle involves the supposition of auricular dilatation. It is indeed possible that without insufficiency of the mitral or tricuspid valves, an impulse may be produced by the momentum communicated to the blood contained within a dilated and distended auricle by the backward pressure of these valves during the ventricular systole.

It is to be borne in mind that the occurrence of movements in the intercostal spaces, impulsive or retractive, involves contingencies irrespective of cardiac disease. They are more likely to occur in persons who have flattened chests and long sternums than in those with a thoracic conformation the opposite of this. They require a certain thinness of the parietes of the chest, and are more marked in proportion as the thoracic walls are attenuated. They may be obvious when the heart is excited, and not appreciable when the organ acts feebly. They may be due to abnormal conditions pertaining to the lungs, the heart remaining sound. They are observed in some instances in which the pulmonary substance is withdrawn from the heart, as after the absorption of liquid effusion in pleurisy affecting the left side and in some cases of tuberculosis. An effect of these affections is often to leave an enlarged area of the heart's surface in contact with the walls of the chest, and, under these circumstances, the motions of the organ may communicate corresponding movements to the intercostal spaces. Hence, impulsive movements elsewhere than over the apex of the heart are never signs of enlargement, unless associated with altered situation of the apex-beat and other signs indicating that the bulk of the organ is increased.

The conformation of the chest in some persons is such that an impulse referable to the heart is felt, in health, in the epigastrium by directing the fingers upwards and outwards beneath the false ribs on the left side. In the majority of persons the organ is too

[1] On Diseases of the Heart and Aorta, Am. ed., p. 290. Dr. Stokes attributes the impulse over the dilated auricle, in that case, to the auricular contraction; but as he states that it was synchronous with the ventricular systole or the first sound of the heart, it seems clearly to have been due to a regurgitant current.

far removed for its action to be appreciable in this situation. Cardiac impulse in the epigastrium is therefore usually, but not invariably, a sign of disease. As a morbid sign, it denotes either enlargement of the heart or displacement in a downward direction. It is a sign by no means present in most cases of enlargement of the heart. The oblique position of the heart and the resistance offered by the diaphragm and the left lobe of the liver prevent much descent towards the epigastrium. These circumstances apply, as has been seen, measurably, to cases in which the enlargement predominates in the right as well as the left ventricle. But it is undoubtedly true that an impulse in this situation is more likely to occur as a result of enlargement of the right than of the left ventricle. When it proceeds from a cardiac affection, it may be considered as affording strong presumptive evidence that the right ventricle is enlarged. A strong impulse, under these circumstances, goes to show that the enlargement involves not merely dilatation, but hypertrophy. The question to be first settled is, Does it proceed from increased size of the heart? This question may be settled frequently by reference to the apex-beat. If the beat be in its normal situation, and there are no signs of enlargement, the impulse in the epigastrium is probably normal. It is not a sign of disease. But if the apex-beat be removed to the left of its normal position, it becomes a sign of enlargement of the right ventricle. When this is the case, other signs of enlargement will also be present. The diagnostic value of the sign, thus, when it is attributable to a cardiac affection, consists in its indicating that the right ventricle is the seat of enlargement. When it is determinable that the epigastric impulse is due to cardiac enlargement, the extent of the impulse will, in some measure, be an index of the amount of increase of the bulk of the right ventricle, and the power of the impulse will be in proportion as the enlargement is by hypertrophy, provided that the organ is not weakened from any cause, or prevented from contracting completely. The impulse is communicated in some instances not only to the epigastrium, but to the lower part of the sternum, and it is sometimes sufficient to cause a movement perceptible to the eye and touch, which extends over the site of the liver. When due to displacement of the heart, in the great majority of instances it is dependent on emphysema affecting the left lung. The dilated lung presses the heart downwards, overcoming the resistance offered by the diaphragm and liver, and the action of the right ventricle is felt in the epigastrium. The signs and symp-

toms of emphysema are sufficient to establish the fact that this cause of displacement exists. The apex-beat, under these circumstances, is frequently or generally wanting. Emphysema, however, induces enlargement of the heart, seated primarily and especially in the right ventricle. The epigastric impulse, therefore, may be due to both causes combined, viz., enlargement and displacement. To determine the proportion which each bears in the production of the sign is not easy. If the boundaries of the heart are determinable by percussion, or if the situation of the apex-beat can be ascertained, this point may be settled with much precision. In examining the epigastrium with reference to the evidence of cardiac enlargement, it is important not to confound an impulse undoubtedly referable to the heart with pulsations often felt in that situation which are only indirectly attributable to the heart's action. In some thin persons, the beating of the descending aorta may be here felt; and in connection with hysteria and other nervous affections, especially when accompanied by gastric tympanites, strong pulsations are perceived in the epigastrium, which are said not to be uniformly synchronous with the heart's movements, and the mechanism of which it is not easy to explain. It is not difficult by means of palpation either to trace these pulsations directly to the heart or to isolate them from the latter.

The action of the heart is frequently attended by a shock felt by the hand or the head applied over the præcordia. Sensible movements are also sometimes communicated to the ribs, as well as the intercostal spaces, and they may extend over the præcordia. When the heart is tranquil, in health, a shock is rarely if ever perceived. The fifth rib is occasionally slightly raised by the movements of the apex during the systole. In disease, these effects of the heart's action are often marked. A perceptible and more or less forcible shock attends certain palpitations which are merely functional. The heart appears to act with violence. It seems to knock against the ribs. The sensation, in some instances, is as if the chest were struck with a hammer. The patient is painfully sensible of the force of the impulsion, while, in health, if the heart be not excited, its movements take place without the mind being cognizant of them. The violence of the action is shown by the movements of the body, of the dress, of the bedclothes. The instances related of fracture of the ribs and detachment of the costal cartilages by the force of the heart's action are doubtless apocryphal, but the shock is sometimes very great. It may be limited to the apex or felt at

the base, and, indeed, over the whole præcordia. Alone, the shock, however violent, only indicates excited action of the heart. It does not, of necessity, imply organic disease. It may be due simply to the fact that the heart acts with spasmodic or convulsive quickness and force. It is represented by the intense action incident to fear and some other emotions. If it be inorganic or functional, it is usually temporary, unattended by physical signs denoting organic lesions, and characterized by circumstances which will be hereafter considered as distinctive of nervous disorder or palpitation. Organic disease, it is true, is often attended by violent action of the heart, but the significance of the latter as a sign of the former depends on the coexistence of other signs which are more unequivocal; and, on the other hand, organic disease is often present when the heart's action is more feeble than in health. A strong heaving movement of the ribs or the præcordia is, however, highly significant of enlargement by hypertrophy. This is quite different from the shock which has just been described. It is a comparatively sluggish, prolonged, powerful elevation of the thoracic walls. The head, applied as in immediate auscultation, is raised, and, by the hand placed over the præcordia, the heart is felt to act with abnormal strength. The shock, due to intense functional excitement, proceeds merely from exaggerated action of the heart; the heaving movement in hypertrophy involves, in addition, increased power of the muscular contractions of the organ. Moreover, in the latter case, the surface of the heart being in contact with the thoracic walls over a larger area, the extent of the impulsive movement is greater. The distinction just drawn is the same as has been already pointed out in contrasting the prolonged, powerful apex-beat of a hypertrophied heart with the smart, sharp, violent impulse which only indicates excited activity of the ventricular systole. The distinction in both instances is practically important, but in discriminating between functional disorder and organic disease, in practice, the diagnostician will, of course, be guided by the absence or concurrence of other signs. It is hardly necessary to state that heaving of the præcordia is not uniformly present in hypertrophy. The presence of this sign involves, as a condition, a degree of functional activity proportionate to the augmented thickness of the ventricular walls; in other words, it will not be present if the muscular power of the heart be weakened from any cause, notwithstanding the augmented bulk of the organ. Clinical observation, in fact, shows that a heaving impulse is often

wanting in cases of hypertrophy. While, therefore, the presence of this sign is evidence of the existence of hypertrophy, its absence is by no means proof that hypertrophy does not exist.

3. *Abnormal modifications of the heart-sounds; diminished extent and degree of the respiratory murmur and of vocal resonance within the præcordia, as determined by auscultation.*

The clinical importance of abnormal modifications of the heart-sounds has relation more to valvular affections than to enlargement of the heart. They are, however, by no means unimportant in connection with the latter. And here, as in treating of the physical signs embraced in the two classes already considered, it will be necessary to premise some account of the heart-sounds in health. To enter into a discussion of the numerous theories which have been advanced with regard to the mechanism of these sounds, would be tedious and unprofitable, as well as foreign to the practical character of this work. I shall limit myself to a concise statement of points which are essential as preliminary to the study of the phenomena of disease; and I shall devote to these less space in consequence of having recently considered them in a special publication, to which the reader is referred for a fuller exposition of the subject.[1]

The two heart-sounds, which together form the beat or revolution of the heart, are called the first and second, or the systolic and diastolic sound. By the latter terms, it is implied that the first sound occurs during the systole and the second sound during the diastole of the ventricles. This, although called in question by some, may be assumed as sufficiently established.[2] These sounds, respectively, have their maximum of intensity, and their characters are best studied in different situations, viz., the first sound over the point where the apex-beat is felt, and the second sound just above the base of the heart, in the intercostal space between the second and third ribs near to the sternum. Studied in these different situations, the two sounds differ as respects duration, pitch, and quality. The first sound, over the apex, is longer, lower, and has

[1] On the Clinical Study of the Heart-Sounds in Health and Disease. Prize Essay. Transactions of the American Medical Association, vol. xi., 1858.

[2] M. Beau contends that the first sound is due to the auricular contractions. Op. cit.

a booming[1] quality. The second sound, in the second intercostal space near the sternum on either side, is shorter, more acute, and has a flapping or valvular quality. These differences in characters between the two sounds are generally well marked when the comparison is made in the different situations mentioned, but, as will be seen presently, they are much less marked in other situations within the præcordia. The sources of each of the sounds, and the parts concerned in its production, are important to be considered. With reference to these and other points, we will notice each sound separately, commencing with the second sound.

The second sound succeeds the first after an interval extremely brief, but, when the beats of the heart are not much accelerated, distinctly appreciable. It is estimated that the duration of this interval and the second sound combined is equal to that of the first sound, or of the longer interval which separates the second sound from the succeeding first sound, the latter interval and the first sound being about equal in duration. This sound, *i. e.*, the second, as already stated, is best studied just above the base of the heart, in the space between the second and third ribs, near to the sternum. If the second sound be compared on the two sides of the sternum, a difference in pitch and other characters is generally apparent. On the right side the sound is more acute, more abrupt, louder, and apparently nearer the ear. These differences, taken in connection with the anatomical relations of the aorta and pulmonary artery in these situations, and also with clinical facts pertaining to disease, warrant the conclusion that, when a disparity exists, the sound on the left side emanates from the pulmonary artery, and that on the right side from the aorta. The sound in both situations has an unmixed, valvular quality, and, in view of the results of experiments made on living animals, and the effects of disease, it may be assumed that the valves of the aorta and pulmonary artery are the parts immediately concerned in its production. There is, then, a pulmonary second sound, due to the expansion of the valves of the pulmonary artery succeeding the ventricular systole, and an aortic second sound, referable to the semilunar valves of the aorta. The second sound of the heart presents the characters of that due to the pulmonary valves, at the inferior border of the organ, *i. e.*, just above the xiphoid cartilage in some persons; occasionally, also, in

[1] The term booming, borrowed from Dr. Walshe, has not a very definite signification; but expresses a difference in quality difficult to be described, although easily appreciated by the ear.

the third intercostal space on the left side and over the body of the heart, within the superficial cardiac region. Elsewhere, within the præcordia, and at points removed from the præcordia, wherever the second sound is heard, it presents the characters distinctive of the sound produced at the aortic orifice. These facts are ascertained by comparing, in a sufficient number of healthy persons, the second sound, as heard at different points, with that heard in the second intercostal space on the right and left side. It follows from the facts just stated that the aortic second sound is much more intense and widely diffused than the pulmonary, the latter, in some persons, being distinguishable alone in the second intercostal space on the left side; sometimes, indeed, the aortic sound predominates even in that situation. The second sound of the heart maintains its distinctive characters of pitch, duration, and valvular quality, unaffected by the causes which affect the movements of the heart within the limits of health, such as exercise, mental agitation, etc. Its intensity even is not much affected by these causes. These facts show its unmixed character, in other words, that it consists of a single element only, a valvular element, in this respect differing from the first sound of the heart.

The first sound of the heart, studied at the situation where its intensity is greatest, viz., over the apex of the organ, is a mixed sound. In this situation, it is usually *accentuated*, that is, in the succession of the two sounds the stress falls upon the first, while at the base of the heart, and at other points, the accent is on the second sound. The mixed nature of the first sound is shown by the difference which it presents on auscultation over the apex, and at other points within the præcordia; by contrasting its characters as heard when the stethoscope is firmly placed directly on the surface of the chest with those which it presents when some soft material is interposed between the instrument and integument, or when the instrument is imperfectly applied; by auscultating over the apex when the person examined is placed in different positions, and taking into consideration modifications incident to certain diseases and peculiar to certain persons in health.[1] The clinical study of this sound in health and disease leads to the conclusion that it is composed chiefly of two different elements. One of these elements consists of a valvular sound, due to the action of the

[1] In the prize essay already referred to (*Trans. Am. Med. Association*, vol. xi., 1858), the author gives a full account of the results of the clinical study of this sound under the different circumstances mentioned above.

mitral and tricuspid valves. The other element, in the author's opinion, proceeds from the movement of the apex of the heart against the thoracic walls. In a practical point of view, however, it is unimportant whether the latter element be thus explained or whether it be accounted for on the hypothesis of a sound adequate to its production, due directly to muscular contraction. Referring it to the movement of the apex against the thoracic walls, this element may be called the *element of impulsion*, and the other element the *valvular element*.[1] These names will be employed in this work to distinguish from each other the two elements composing the first sound.

These two elements of the first sound are combined in different proportions in different situations in which auscultation is practised, in different positions of the body, and under different circumstances pertaining to disease. At certain points, the element of impulsion may be eliminated, leaving the valvular element alone present. The element of impulsion predominates and drowns the valvular element, often on auscultation over the apex. It predominates, as a rule, over the body of the heart. At the base of the heart the valvular element frequently predominates. At the left border of the heart, over the left nipple, the valvular element predominates, and, on carrying the stethoscope to the left of this point for a greater or less distance, the element of impulsion is eliminated, and the valvular element remains, leaving the sound as purely valvular in quality and as short as the second sound. The valvular element predominates generally at the right border of the heart and at all the points removed from the præcordial region where the first sound is appreciable. These facts, established by the clinical study of the heart-sounds in health, show that, although the element of impulsion predominates over the apex and body, the valvular element alone is much diffused beyond the limits of the organ.

[1] Discussion of this opinion, respecting the mechanism of the element of impulsion of the first sound, is waived in this work. The reader is referred to the author's prize essay on the clinical study of the heart-sounds for the grounds on which the opinion is entertained. I will simply add here that the experiment of placing Cammann's stethoscope over the naked heart, when exposed in a living animal, seems to me sufficient to disprove the hypothesis that muscular contraction furnishes an element of the first sound. The first sound in this experiment is intensely valvular. This sound sometimes has a similar intense valvular quality, in cases of great functional excitement of the organ, when the stethoscope is applied on the chest over the point of apex-beat, the element of impulsion being, from some cause, wanting.

The valvular element is less intense than the second sound, the latter being often heard in situations to which the former is not transmitted, viz., on the lateral surfaces of the chest, in the right infra-clavicular region, and over the back.

The valvular element of the first sound, as stated already, is due to the action of the mitral and tricuspid valves. Is the sound emanating from each of these valves ever distinguishable from the other? Clinical observation warrants an affirmative answer to this inquiry. Over the inferior border of the heart, near the xiphoid cartilage, this element frequently differs in pitch from the same element when heard in the same person at or without the left nipple. This may be considered as sufficient to render it at least highly probable that the source of the sound in the latter situation is at the mitral, and in the former situation at the tricuspid valves.

A striking point of disparity between the first and second sound of the heart relates to the extent of variation in intensity in different persons, and in the same person under different circumstances within the limits of health, as well as in connection with disease. The first sound varies considerably in intensity according to the energy with which the heart contracts, and according to the posture assumed; it is often feeble when the person lies on the back as compared with intensity in the sitting posture, or lying on the left side. The second sound, on the other hand, undergoes little change in intensity under these and other circumstances, irrespective of morbid conditions. The variation to which the first sound is liable relates chiefly to the element of impulsion. The valvular element, like the second sound of the heart, is not subject to much variation in intensity, exclusive of disease.

The relatively greater duration of the first sound of the heart, as compared with the second sound, depends on the element of impulsion. In proportion as this element is predominant is the sound prolonged; and, on the other hand, whenever this element is eliminated, the first sound is no longer than the second. The interval between the first and second sound is determined by the length of the first sound. This interval is shortened in proportion as the first sound is prolonged, and it is lengthened in proportion as the element of impulsion of the first sound is impaired or eliminated.

The foregoing brief account of the heart-sounds in health embraces, as concisely as possible, the more important of the conclu-

sions deduced from the results of the analysis of the phenomena obtained by auscultation in the examination of twenty-five persons presumed to be entirely free from disease, the phenomena being carefully noted at the time of the examination. For a fuller account of these results, the reader is referred to the publication already alluded to. It remains now to notice the modifications of the heart-sounds observed in connection with hypertrophy of the heart. The modifications significant of hypertrophy differ materially from those which pertain to dilatation. The former relate to the present subject. The latter will be noticed in another section in connection with enlargement by dilatation.

Hypertrophy of the left ventricle tends to exaggerate the element of impulsion of the first or systolic sound so long as the muscular power of the heart remains unimpaired. The impulsive movements of the apex against the walls of the chest, *cæteris paribus*, are proportionate to the hypertrophy of this ventricle. Exceptions to this rule occur when the form of the organ is so changed that the apex ceases to come into contact with the thoracic walls, or when, owing to muscular weakness, the impulsive movements are diminished instead of being increased. All observers have remarked that in cases of hypertrophy, while the muscular energy of the heart is proportionate to its increased bulk, the first sound is notably dull and prolonged. The dulness and prolongation of this sound, as compared with the second, in health, are due to the element of impulsion. It is, therefore, quite intelligible that when the impulsive movements are increased, the effects on this sound are abnormal dulness and prolongation, as well as exaggerated intensity. Mere exaggeration of this sound is by no means in itself significant of hypertrophy. Increased muscular action of the heart, as in some instances of functional disorder, renders the sound abnormally intense, so that it is sometimes appreciable at a distance from the chest, and painfully perceived by the patient. Both elements of the sound, under these circumstances, are exaggerated. This is also true in cases of pure hypertrophy, *i. e.*, uncomplicated with valvular lesions; but in hypertrophy the element of impulsion is relatively more exaggerated than the valvular element, and hence, when the dulness and prolongation are marked, as well as the increased intensity, the modification becomes significant of this affection. Modifications affecting the valvular element of the first sound are of importance chiefly in connection with the diagnosis of

valvular lesions. The modifications significant of hypertrophy relate more especially to the element of impulsion.[1]

Modifications of the second or diastolic sound, incident to hypertrophy, may affect the aortic and the pulmonary sound separately or combined. The pulmonary and the aortic sound are in relation respectively to the right and left ventricle. The expansion of the semilunar valves succeeding the ventricular systole is due, in a great measure at least, to the systolic contraction of the ventricles. The column of blood propelled from the ventricles dilates the aorta and pulmonary artery, and the recoil due to the elasticity of the coats of these vessels during the ventricular diastole gives rise to the expansion of the valves, which occasions the second sound. This is the explanation now generally received of the mode in which the expansion of the valves is produced. Whether another agency be not involved in the production of the second sound, viz., an active diastolic expansion of the ventricles, is a matter of question. The force derived from the elasticity of the arteries, if not the sole agency, is, at all events, the most important in causing the expansion of the valves. This force, it is obvious, other things being equal, is proportionate to the power of the ventricular systole. The dilation of the aorta and pulmonary artery is greater the more powerful the contractions of the ventricles, and the rebound of the arterial coats is stronger the more the vessels have been dilated. Hence, the intensity of the second sound of the heart represents the power of the systolic contractions of the ventricles; and the aortic and the pulmonary sound respectively represent, in this respect, the left and the right ventricle. The two ventricles, as has been seen, may become enlarged by hypertrophy separately, as well as conjointly; and when both are affected the enlargement of one generally predominates over that of the other. It might, therefore, be expected, and clinical observation shows that an abnormal intensity of the aortic and the pulmonary sound sepa-

[1] My clinical observations have led me to regard exaggeration of the tricuspid portion of the valvular element of the first sound as evidence, in some cases, of hypertrophy of the right ventricle. To determine the fact of its exaggeration, the valvular element of the first sound is to be compared at the inferior boundary of the heart, near the xiphoid cartilage, with this element at the left border of the heart at or without the left nipple. In health, this element of the first sound is notably more feeble in the former than in the latter situation. If the valvular sound be equally or more marked at the inferior boundary of the heart, provided the mitral valves are sound, it is evidence that hypertrophy of the right ventricle exists, if other signs of cardiac enlargement are at the same time present.

rately, may become a sign of hypertrophy affecting, in the one case, the left, and, in the other case, the right ventricle.

Hypertrophy of the left ventricle gives rise to exaggerated intensity of the aortic second sound, *i. e.*, the sound having its maximum of intensity in the second intercostal space on the right side of the sternum, provided this effect be not prevented by attendant circumstances, which are of frequent occurrence. Lesions affecting the aortic valves, diminished elasticity of the aorta from disease of its coats, contraction at the mitral orifice, or mitral regurgitation, both lessening the column of blood propelled by the ventricle into the aorta, are circumstances which obviously stand in the way of an abnormal increase of the aortic second sound proportionate to the augmented power of the ventricle. Hypertrophy of the left ventricle is seldom altogether devoid of these circumstances. In point of fact, it is only in the rare instances of uncomplicated hypertrophy of this ventricle that the aortic second sound is notably exaggerated. As a physical sign, therefore, it has very little value.

Hypertrophy of the right ventricle, on the other hand, is seldom associated with circumstances preventing its effect on the pulmonary second sound, *i. e.*, the sound as heard in the second intercostal space on the left side of the sternum. Lesions of the semilunar valves of the pulmonary artery, and of the tricuspid valves, are of extremely infrequent occurrence. Exaggerated intensity of the pulmonary second sound, therefore, is highly significant of hypertrophy of this ventricle. This effect is especially marked if, in conjunction with increased power of the ventricular contraction, there exists congestion of the pulmonary vessels involving obstruction to the free passage of blood through the lungs. The resistance which the column of blood propelled into the pulmonary artery meets with, induces a greater dilation of this artery during the ventricular systole, and, consequently, a stronger recoil after the systole, giving rise to a louder pulmonary second sound. Pulmonary congestion, often due to mitral contraction or regurgitation, generally co-exists with hypertrophy of the right ventricle, and stands to the latter in the relation of causation. In estimating the amount of exaggerated intensity of the pulmonary second sound, it is to be compared with the aortic second sound in the same intercostal space on the right side of the sternum. In making this comparison, it is to be borne in mind that lesions affecting the mitral orifice (contraction, or regurgitation, or both), which are

often associated with hypertrophy of the right ventricle, involve diminished intensity of the aortic sound by lessening the amount of blood propelled by the contraction of the left ventricle into the aorta. Under these circumstances, the pulmonary second sound may be more intense than the aortic, when its actual intensity is not augmented. Exaggeration of the pulmonary second sound occurring in connection with the mitral lesions just named, will be again noticed in treating of these lesions. It is also to be borne in mind that in mere functional excitement of the heart, both the pulmonary and aortic second sound acquire an abnormal intensity. Under these circumstances, the second sound, in both situations, is alike exaggerated. Abnormal increase of the intensity of the sound emanating from either the aorta or pulmonary artery, is more significant of hypertrophy than when the sound from both of these sources is alike augmented. But with respect to the second, as well as the first sound, abnormal increase of intensity is to be considered as a sign of hypertrophy only when other physical signs of enlargement of the heart are at the same time present. Another point is not to be lost sight of, viz: In the progress of hypertrophy, a period arrives when the muscular power of the heart becomes abnormally weak, notwithstanding the increased thickness of the muscular walls. When this period arrives, the heart-sounds are feeble in proportion to the weakness of the ventricular contractions.

Enlargement of the heart gives rise to certain abnormal changes as regards the respiratory murmur and vocal resonance within the præcordia, which possess some importance as physical signs. In health, the respiratory murmur may, or may not be perceived within the superficial cardiac region during tranquil breathing; but it is generally heard everywhere within the præcordia when the breathing is forced. In cases of enlargement, however, in which the area of the superficial cardiac region is increased, not only is the murmur in tranquil breathing inappreciable, but it may not be discoverable although the breathing be forced. This is corroborative of the more reliable evidence of enlargement afforded by percussion and palpation. The vocal resonance, in health, when more or less marked over the left side of the chest, is either extinct or notably diminished within the præcordial region. The boundaries of the heart may often be as accurately defined by auscultating the voice as by percussion; and, in conjunction with the latter method, the former may be resorted to in determining the augmented space which the heart occupies in cases of enlargement.

4. *Enlargement of the præcordia and abnormal movements, as determined by inspection.*

In healthy persons, free from spinal curvature and obvious deformity of the chest, the præcordial region and the corresponding section on the right side do not present any marked deviation from symmetry. On close comparison with the eye, frequently a slight disparity is perceived, one side projecting a little more than the other. Of the instances, according to my observations, in which this disparity is perceptible, the right and the left side are found to project in an equal proportion. Of twenty-five examinations of different persons in health, with well formed chests, and no spinal curvature; in seven, no disparity was observable; and in an equal number, viz., in nine, the right and the left side, respectively, were found to be slightly more prominent. Three of these persons were left handed. In one of these three persons, the right side was more prominent; in another, the left side, and in one there was no disparity.[1]

Abnormal prominence of the præcordial region occurs in certain cases of enlargement of the heart. The prominence is considerable in some cases, when the heart is enlarged in early life. In a moderate amount, it is not uncommon in cases in which the affection is developed after adult age. Præcordial prominence, due to the accumulation of liquid within the pericardial sac, in cases of pericarditis, may generally be distinguished from that due to enlargement of the heart, by characters determinable by inspection, although the differential signs obtained by other methods of exploration are more strongly marked. The shape of the præcordial projection is not the same in enlargement of the heart as in pericarditis with effusion. In the latter it extends more in a vertical than in a transverse direction. In the former, the arching is wider, and does not extend much, if at all, above the normal situation of

[1] M. Woillez found, of 197 subjects in good health, and without spinal curvature, that in 47 only was the symmetry absolutely perfect. A projection of the left side, in front, either at, or above, or below the nipple, existed in the proportion of 26 per cent. An anterior projection of the right side existed in only two instances. The proportion of instances in which deviation from absolute symmetry existed in my comparatively few examinations, agree very nearly with those of M. Woillez. The proportion of instances in which prominence of the left side was noted is larger in my examinations, and the relative number of instances in which prominence of the right side was observed, is still greater.

the base of the heart. Præcordial prominence due to enlargement, if it exist in a notable degree, denotes both hypertrophy and dilatation, because it is in this species of enlargement that the heart attains to a large size. The projection is very rarely, if ever, so great as in certain cases of chronic pericarditis. The intercostal depressions are not so uniformly abolished. Bulging of the intercostal spaces, which may result from pericardial effusion, never occurs in cases of enlargement. Widening of the intercostal spaces does not take place to the same extent in cases of the latter as of the former. In enlargement, the apex-beat is generally seen and felt, while in pericarditis it is often suppressed; and if appreciable in the latter affection, it is raised above its normal position, while in the former it is often lowered and carried to the left. Other points of distinction will be noticed in treating of pericarditis. It may be added here that the prominence dependent on enlargement is permanent and unchangeable, while that due to pericardial effusion is sometimes developed under the eyes of the practitioner, and, after variations at different times, may finally disappear and be followed by depression.

Movements of impulsion and retraction referable to the heart in cases of enlargement, which have been considered in connection with palpation, are, in general, appreciated by the eye as well as by the touch. Retractive movements may be ascertained by inspection when they are not perceived by palpation. The retraction of the apex-beat is sometimes plainly seen, when an impulse cannot be felt. The alternate movements in different intercostal spaces, which were described as determined by palpation, are best ascertained by inspection. The applicability of this method of exploration to the study of the movements communicated by the heart to the thoracic walls, is to be borne in mind, but it is needless to repeat in this connection the account of these movements, which has been already given.

5. *Increased size of the chest as determined by mensuration.*

The value of mensuration in cases of enlargement of the heart, consists in its giving exactitude to certain of the signs obtained by inspection. It is not essential to the development of data for diagnosis.

As regards measurements of the healthy chest, with reference to the præcordia, the following are the conclusions deduced from

twenty-five examinations in which the circumference was measured with graduated inelastic tape, and the diametrical distance by means of callipers. Equality of the two sides of the chest, and a greater size of the left side, as regards circumference and antero-posterior diameter, do not alone constitute evidence of cardiac or other intra-thoracic disease. This statement holds good within certain limits; in other words, greater size of the left than of the right side beyond half an inch, either by diametrical or circular measurement, points to the existence of disease. Diametrical measurement gives a larger number of instances in which the two sides are equal, than circular measurement, the ratio being six to eleven. The right side was greater in eleven instances as measured by the tape, and in seven as measured by callipers. A greater size of the left side existed in an equal number of instances as determined by the tape and callipers, viz., in five. In all of sixteen cases in which diametrical measurement showed greater size of either the right or left side, the same results had been previously obtained by inspection, with a single exception.

Thus, in confirming and giving greater exactitude to the results of inspection, as respects the size of the chest in cases of cardiac disease, diametrical is to be preferred to circular measurement.

The antero-posterior diameter of the chest at the præcordia is increased in certain cases of enlargement of the heart. In determining that it is due to cardiac disease, abnormal conditions referable to the lungs or pleura, increasing the size of the chest, are to be excluded by the absence of the signs denoting their existence; and the abnormal increase of the diametrical dimension of the left side is referred to an abnormal condition of the heart, not alone by the exclusion of diseases affecting other intra-thoracic structures, but by concomitant signs of cardiac enlargement. The advantage of mensuration as already stated, is mainly in corroborating the evidence afforded by the eye, and in enabling the physician to determine with greater precision the amount of disparity between the two sides. In recording cases, it is more satisfactory to note the results of a comparison of the two sides in figures than to express them in terms which are somewhat indefinite; such as slight, moderate, great, etc. With reference simply to diagnosis in individual cases, inspection suffices without resorting to measurement.

The diagnosis in cases of enlargement of the heart and hypertrophy must rest on the physical signs. The symptoms which

have been mentioned (page 33) may point to these lesions, and afford corroborative evidence of their existence, but they are not adequate to lead to positive conclusions. So far as concerns enlargement, it is determinable with great ease and precision by means of physical signs in the vast majority of cases. To determine whether hypertrophy or dilatation predominate is more difficult, but in most instances it is practicable with due knowledge and care. As regards these two forms of enlargement, the differential diagnosis will be considered under the head of enlargement by dilatation in an after part of this chapter. The signs involved in the diagnosis of enlargement and hypertrophy are fewer and more simple than would appear from the space devoted to the subject in this chapter. The subject would here require comparatively brief consideration had it not been requisite, in this connection, to introduce accounts of the phenomena obtained by physical exploration in health, as the point of departure for studying the phenomena of disease relating not alone to the diagnosis of the affections treated of in this chapter, but to those which are to be subsequently considered. The greater part of the present section has been occupied with facts which belong to physiology rather than pathology. Having been here introduced, it will only be necessary to allude to them hereafter in treating of subjects as preliminary to which they are equally important. For the convenience of the reader, a recapitulation of the physical signs of enlargement and of hypertrophy is given in the summaries which follow.

SUMMARY OF THE PHYSICAL SIGNS OF ENLARGEMENT OF THE HEART.

1. *Percussion.*—The area of the superficial cardiac region extended beyond the range of healthy variation, especially in width. The degree of dulness within this area greater than in health, and the sense of resistance more marked. The limits of the deep cardiac region, in other words, the boundaries of the heart, generally defined by careful percussion, the dimensions of the space which the heart occupies being thus ascertained with precision, and the form of the organ delineated on the chest. Enlargement of the right or left auricle sometimes determined by the extent of the area of dulness at the base of the heart on the right or left side of the sternum.

2. *Palpation.*—The apex-beat removed to the left of its normal position, and often lowered. The area within which the apex-beat is felt, extended beyond the range of health. Abnormal impulses felt in two, three, and sometimes even four intercostal spaces; the additional impulses either synchronous or alternating with the apex-beat, in some instances referable to the auricles, although due to the ventricular systole; and, when felt in the epigastrium, due to the action of the right ventricle.

3. *Auscultation.*—The respiratory murmur not appreciable within the superficial cardiac region in tranquil breathing, and sometimes wanting when the breathing is forced; feeble over a larger area within the præcordia than in health. The boundaries of the heart defined by abrupt cessation or notable diminution of vocal resonance, and the augmented space which the organ occupies, in this way determinable in corroboration of the evidence afforded by percussion.

4. *Inspection.*—Abnormal projection of the præcordial region in some cases; the projection considerable if the enlargement take place in early life. The movements of impulsion determined, which are also ascertained by palpation; movements sometimes seen which are not perceptible to the touch, especially movements which commence by depression with the systole of the ventricles. Alternate movements of intercostal spaces often apparent to the eye, which are imperfectly ascertained by palpation.

5. *Mensuration.*—Prominence of the præcordia greater than the corresponding portion of the chest on the right side; in some cases apparent on inspection, but determined with precision by diametrical measurement. Mensuration also employed in determining with accuracy the dimensions of the superficial and deep cardiac regions, the position of the apex-beat relatively to the nipple, the median line, etc.

SUMMARY OF PHYSICAL SIGNS DISTINCTIVE OF ENLARGEMENT BY HYPERTROPHY.

1. *Palpation.*—Abnormal force of the apex-beat, denoting not merely excited action of the heart, but augmented power of the systole of the left ventricle, the impulsion prolonged, sluggish, and strong. A strong impulse in the epigastrium in cases of hyper-

trophy of the right ventricle; the impulsions sometimes communicated to the lower part of the sternum, and extending more or less over the site of the liver. A strong, heaving movement of the ribs or the entire præcordia, in distinction from the shock, more or less violent, due merely to augmented functional activity of the ventricles.

2. *Auscultation.*—Exaggeration of the aortic second sound, and especially of the element of impulsion of the first sound, in hypertrophy of the left ventricle, rendering the first sound dull and prolonged, as well as abnormally intense. Exaggerated intensity of the pulmonary second sound, in hypertrophy of the right ventricle, especially if associated with obstruction to the pulmonary circulation. Augmentation of the tricuspid valvular element of the first sound in some cases of hypertrophy of the right ventricle.

Treatment of Hypertrophy.

False notions of the pathology of hypertrophy have hitherto led to erroneous principles of treatment, which govern, still, the practice of very many, if not most physicians. The object has been to devise the most effective means of diminishing the state of hypertrophy, *i. e.*, of reducing the size of the ventricular walls, and, if this be not practicable, of preventing, if possible, progressive increase of the muscular tissue. For this end, some years age, copious and repeated abstractions of blood were employed, in conjunction with low diet, after the plan of Valsalva and Albertini, Italian physicians. This method was found to be pernicious, but, instead of being discarded, the same plan, not carried to the same extent, was recommended by Hope, Bouillaud, and others, and has been generally pursued up to the present time. A better understanding of the pathological relations of hypertrophy leads to the conclusion that therapeutical measures designed to diminish or prevent it, are likely to do harm in so far as they have efficiency in promoting these ends. Considered in connection with the antecedent morbid conditions which give rise to it, conditions involving impediment to the circulation, hypertrophy, so far from being an evil, is an important provision against the dangers incident to accumulation of blood within the cavities of the heart, and against the evils of dilatation, the latter being much the more serious of the two forms of

enlargement. In the great majority of cases, enlargement of the heart is the result of valvular lesions. These lesions often exist for a long time before they give rise to symptoms which lead the patient to suppose that he is affected with disease. When cases first come under the notice of the practitioner, it is evident that the enlargement has been going on for months or even years. The amount of enlargement, when the chest is for the first time examined, shows that it is not of recent production. We have seen that, as regards hypertrophy and dilatation, which are almost always combined, the former, as a rule, takes precedence in time. The hypertrophy, in short, compensates, during a greater or less period, for the disturbance of the circulation caused by the valvular lesions; and so long as the enlargement consists of this compensating increase of muscular structure, and consequently of muscular power, the patient experiences little or no inconvenience, provided nothing occurs, like anæmia, for example, to weaken the force of the heart's action. It is when the hypertrophy has reached the limit of compensation, and dilatation has followed, that serious inconveniences, referable to the heart and circulation, begin to be felt. With this general view of the pathological character of hypertrophy, the indications for treatment may be embraced in three classes, viz: 1. To prevent or limit, as far as practicable, impediment to the circulation dependent on valvular lesions or other conditions, and giving rise to hypertrophy; 2. To obviate, as far as possible, weakness of the heart, and a tendency to dilatation; 3. To quiet undue excitement and irregular action of the heart.

The antecedent pathological conditions giving rise to cardiac enlargement, viz., valvular lesions, pulmonary emphysema, etc., are not of a nature to admit of removal. The physician, however, can do something towards preventing or limiting the impediment to the circulation, which is the immediate effect of these conditions, and which is the intervening cause of enlargement. This indication is fulfilled by avoiding extrinsic causes which excite unduly the action of the heart, by measures designed to equalize the circulation, and by the judicious employment in some cases of bloodletting and other means of depletion. Excessive muscular exercise is objectionable, but, as will be seen presently, within certain limits it is not to be prohibited, but enjoined. Excesses in eating and in the use of stimulating drinks are to be avoided. Mental excitement belongs in the same category. The circulation is equalized by securing, as far as may be, for the different, and especially the

remote parts of the body a proper proportion of blood, thus preventing its undue accumulation within the cavities of the heart. For this end, the surface of the body should be guarded against the influence of cold, and revulsive measures, such as warm and stimulating pediluvia, frequently resorted to if the circulation in the extremities be sluggish. Constipation, if it exist, claims appropriate remedies. Bloodletting is permissible when there exists over-repletion of the general vascular system, the object being, by lessening the mass of blood, to facilitate its circulation. This object should be clearly understood. It is easy to understand that if the vessels are abnormally full of blood, an irremediable impediment to the circulation is likely to occasion greater accumulation in the heart and its cavities than when the mass of blood to be circulated does not exceed the normal amount. The existence of plethora furnishes the indication for bloodletting, and the removal of this state constitutes the limit to which it may with propriety be carried.[1] Carried beyond this limit, the detraction of blood can hardly fail to be pernicious. It is to be borne in mind that bloodletting is not to be practised because hypertrophy exists, but because over-repletion of the vascular system, added to an existing permanent impediment to the circulation, increases the necessity, as it were, for the production of hypertrophy. Injudiciously practised, bloodletting is injurious in proportion as it impoverishes the blood and weakens the muscular power of the heart. Resorted to with reference to the object just stated, it is indicated in only a certain proportion of cases, and the abstraction of a large quantity of blood is very rarely, if ever, called for. The end for which bloodletting is employed may generally be fulfilled by other methods of depletion which involve less risk of doing harm. The use of saline laxatives and diuretics, conjoined with a somewhat restricted diet, and, more especially, with restriction in the quantity of fluid ingesta, will, in most instances, accomplish the object. These means are to be preferred on account of their being free from the evils attending the spoliative effects of bloodletting when employed injudiciously.

The inconveniences arising from hypertrophy are aggravated by

[1] It is assumed that the state of plethora, *i. e.*, abnormal augmentation of the mass of blood, may exist, and also that when the mass of blood is diminished by bloodletting or other means, the vessels are not immediately refilled. The assumption of these points, in opposition to the speculative views of some, is believed by the author to be in accordance with clinical observation.

weakness of the heart. All observers have noticed the evils of coexisting anæmia. Impoverishment of the blood renders the heart irritable, easily excited into violent and irregular activity, while its power of action is impaired. Alarming symptoms are sometimes induced under these circumstances, which are so entirely relieved by restoring the blood to its normal condition that patients imagine themselves completely cured. A patient, rendered highly anæmic by lactation, presented dyspnœa, palpitation, and œdema to such an extent that her condition seemed quite hopeless, but after weaning, the use of tonics, etc., she recovered apparently perfect health, so that, except for the physical signs of cardiac disease, the cure would have been considered complete. Two years afterwards she had apoplexy followed by hemiplegia, which terminated fatally. The combination of anæmia and enlargement of the heart is to be prevented, if possible; and, if it exist, the anæmia, if possible, is to be removed by appropriate measures of medication, diet, and regimen. Irrespective of this condition of the blood, all agencies which tend to weaken unduly the force of the ventricular contractions are contra-indicated. In proportion to the weakness of the heart will be the tendency to dilatation rather than to hypertrophy. The latter is to be promoted, if this be necessary to prevent the former. So long as hypertrophy predominates, the patient is comparatively safe. The inconveniences and dangers are greatly increased in proportion as dilatation succeeds hypertrophy. It is an important object of treatment, therefore, to obviate or retard the tendency to dilatation. With reference to this object, the diet should be nutritious—a substantial, solid diet, adapted to the formation of blood, rich in quality, but not in excess as regards quantity. Muscular exercise within certain limits is to be encouraged rather than repressed. In cases of cardiac disease attended with enlargement, I have been repeatedly struck with the fact that persons engaged in pursuits requiring considerable physical exertions, laborers, mechanics, or active men of business, continue to discharge their duties for a long time without much inconvenience, but fail rapidly so soon as they discontinue their occupations. I am convinced that a certain amount of exercise is not only allowable, but positively beneficial by promoting the heart's vigor and retarding the passage from predominant hypertrophy to predominant dilatation. It will doubtless seem at first strange to many readers that exercise is recommended in cases of hypertrophy, but, while violent exertions, which excite

unduly the action of the heart, are to be avoided, I am satisfied that moderate and even considerable muscular activity conduces to the welfare of the patient.

At the time of writing I can call to mind a number of persons affected with hypertrophy complicated with valvular lesions, who, engaged in active occupations, and pursuing no medical treatment, would be amazed were they fully aware of their pathological condition. I cannot but think that were the nature and extent of the disease clearly explained to these persons, and great quietude enjoined, their chances for tolerable health for a considerable period would be materially impaired. Still less encouraging would be the prospect were they subjected to a course of diet and medication tending to impoverish the blood, reduce the vital forces, and weaken the power of the heart. I cannot avoid the reflection that I have witnessed the injury inflicted by this course of management in not a few cases.

In cases of complicated hypertrophy, the heart is liable to be unduly excited, and irregular action take place, even when extrinsic causes are, as much as possible, avoided. In other words, functional disorder, or palpitation, may be superadded to the organic affections. This is not only a source of inconvenience, but there is reason to believe that the effect is unfavorable as regards the permanent condition of the heart. To quiet undue excitement and irregular action of the heart, is therefore an object of treatment. Certain remedies may be employed with advantage for this object. Digitalis is a valuable remedy, frequently exerting a sedative effect upon the heart, without lessening the power of its action. Under its judicious use, the ventricular contractions often become less frequent, more regular, and apparently more complete. Care is to be taken not to give it in doses sufficient to reduce the pulse much below its normal frequency, and with due care it may be continued for some time without risk of unpleasant consequences. Bouillaud claims that its endermic application, blistering a small space, and sprinkling daily several grains on the blistered surface denuded of its cuticle, possesses great advantages. Others, however, have not observed that the beneficial effects are more marked when this method is employed, than when it is administered internally. It is possible that the same effects may be obtained from the use of the veratrum viride, introduced by Dr. Norwood. Aconite is highly extolled by Dr. Walshe. He gives this the preference over any other remedy in meeting the indication under

consideration. Belladonna is useful in some cases. A belladonna plaster, worn over the præcordia, has seemed to me to exert a decided effect in tranquillizing the heart. The sedative effect of hydrocyanic acid is useful in some cases.

In these remarks on the treatment of hypertrophy, I have not discussed the feasibility of diminishing the abnormal growth of the muscular walls of the heart, a subject concerning which different writers have held opposite opinions. The views of the pathological character of hypertrophy which have been presented, divest this subject of the practical importance which has heretofore been attached to it.

ENLARGEMENT BY DILATATION.

Under this head are embraced, in addition to the rare instances of pure or simple dilatation, *i. e.*, cases in which the capacity of the cavities is increased, and the walls attenuated, all cases in which the relative amount of dilatation exceeds that of hypertrophy. Of the two kinds of enlargement, this is by far the most frequently found after death in the cases in which organic disease of the heart proves fatal. In the instances in which the heart attains to a very large size, dilatation almost invariably preponderates. The cases in which the organ, from its immensely augmented bulk, resembles a bullock's heart (*cor bovinum*), are those in which there exists a great amount of hypertrophy, together with a still larger amount of dilatation. The degree of dilatation varies greatly in different cases, and the lesser amount of hypertrophy combined with it, is also variable. The preponderance of the dilatation, when the heart is examined after death, is generally sufficiently evident on inspection. The abnormal increase in the dimensions of the organ exceeds that of the weight. The ventricular walls collapse, and the organ, resting on its posterior surface, is flattened, instead of preserving a globular form, as when hypertrophy predominates. The greater increase in width than in length, is marked in proportion to the preponderance of dilatation. Owing to this, the organ becomes wedge-shaped, and sometimes presents nearly a square form.

The pathological process involved in dilatation is quite different from that which occasions hypertrophy. In the latter instance, the

process is vital, in the former, mechanical. Hypertrophy is a consequence of over-nutrition; dilatation is the result of the yielding of the walls of the heart to a distending force. The condition, however, which stands immediately in a causative relation to both processes is the same, viz., undue accumulation of blood within the cavities of the heart; hence it is that both processes take place either conjointly or in succession, and that hypertrophy and dilatation are almost invariably associated. Dilatation, thus, not less than hypertrophy, depends on antecedent affections which occasion impediment to the circulation through the vessels or the orifices of the heart, leading to over-accumulation of blood within the centres. These antecedent affections, with which the dilatation is complicated, are the same as in cases of predominant hypertrophy; and the several compartments of the heart become affected singly and in succession, as in the latter form of enlargement. It is not necessary, therefore, in this connection, to consider the dilatation of these compartments, respectively, in relation to the particular lesions of the valves and orifices and vessels on which dilatation and hypertrophy alike depend. Moreover, both dilatation and hypertrophy of the different divisions of the heart will be referred to hereafter in treating of valvular lesions. It will suffice to inquire into the circumstances which determine the occurrence of dilatation in the place of, or, as is generally the case, in addition to hypertrophy.[1]

The first effect of an undue accumulation of blood in the cavities of the heart, continued for a sufficient period, is increased muscular action and consequent hypertrophy in the great majority of cases. The hypertrophy is more or less progressive, but it has its limit. The abnormal growth of the muscular tissue ceases at a certain point. But the morbid conditions inducing over-repletion of the cavities, still remain, impeding more and more the circulation. The compensating increase of the muscular tissue no longer taking place, the walls of the cavities yield to the mechanical force of distension and the progressive enlargement from this time onward is due to dilatation. The limit of hypertrophic enlargement varies in different persons. If it do not cease till the muscular walls attain to a great thickness, and life continue for a long period afterward, the dilatation finally predominates, and the result is an

[1] Of 209 cases of dilatation analyzed by Dr. T. K. Chambers (*Decennium Pathologicum*), in 69 the valves were free from disease, leaving 140 cases of complicated dilatation.

enormous enlargement of the heart, a cor bovinum. But dilatation may commence after moderate or slight hypertrophy has taken place; in other words, the hypertrophy ceases after a smaller amount of muscular growth, and dilatation commences. Dilatation may even commence without any previous hypertrophy, and the result is, then, enlargement with attenuated walls, or simple dilatation, a rare variety of cardiac enlargement. The occurrence of dilatation is determined by the state of the muscular walls. Functional debility of the organ, and, still more, changes in the muscular fibres, prevent that vigorous activity which induces abnormal growth, and yielding of the walls takes place early in proportion as the vital power of resistance is impaired. Anæmia, the feebleness consequent on pericarditis and adherent pericardium, fatty degeneration, softening, and any changes which compromise the muscular power of the organ, tend to abridge hypertrophy and favor dilatation. The latter will therefore predominate in proportion as the condition of the walls is such that they early and readily yield to the distension caused by the accumulation of blood within the cavities. After this brief consideration of the circumstances determining the occurrence of dilatation, in addition to the incidental remarks already made under the head of enlargement by hypertrophy, the reader will be able to trace the relations of dilatation affecting the different cavities of the heart to lesions of the mitral and aortic orifices, involving either obstruction or regurgitation, or both; and to obstructions affecting the pulmonary and systemic arterial systems at situations more or less remote from the heart, without a recapitulation of the account already given in connection with hypertrophy. The inquiry arises, Does not the heart in some instances become dilated in consequence of inherent weakness, no antecedent affections existing to occasion impediment to the circulation? It is probable that this sometimes occurs as an effect of fatty degeneration, pericardial adhesions, atrophy or softening of the muscular fibres, etc. Examples are found of dilatation associated with these structural changes, and without other obvious sources of impediment to the circulation. These changes may take place subsequent to dilatation, but it is reasonable to suppose that in some instances they precede and give rise to it. Clinical observation, however, furnishes no evidence that functional weakness alone leads to dilatation, irrespective of structural changes of the walls of the heart, or lesions of some kind which occasion impediment to the circulation. Dr. T. K. Chambers has suggested that

general obesity may prove a cause of dilatation, in consequence of the "increased area of capillaries through which the blood has to be propelled in fat people."[1]

Symptoms and Pathological Effects of Dilatation.

The symptoms due to dilatation, like those of hypertrophy, are generally so involved with those incident to valvular or other concomitant lesions, that it is difficult, if not impossible, to disconnect them entirely from the latter in individual cases. The materials for the clinical history of simple, uncomplicated dilatation (excluding not only valvular lesions and obstructive affections more or less removed from the heart, but also diseases of the pericardium and structural changes of the cardiac walls), are yet to be collected. An approximation, however, may be made toward the symptomatology of this form of enlargement, by contrasting cases of complicated hypertrophy with those of complicated dilatation. In proportion as dilatation predominates, the power of the heart is impaired. The symptoms distinctive of dilatation, in fact, proceed from feebleness and incompleteness of the heart's action. The action of the heart is often irregular, as represented by irregularity of the pulse and of the apex-beats. Both are abnormally feeble. The pulse may be unequal as well as irregular, but it is difficult to say to what extent this may be owing to concomitant valvular affections. The patient experiences more or less uneasiness and undefinable distress referable to the præcordia, but he is not conscious of that powerful action of the heart which characterizes hypertrophy. Visible throbbing of the superficial arteries is not perceived. The extremities and surface of the body are cool. Lividity may be apparent on the prolabia, the tongue, face, and extremities. The veins may be distended. These symptoms are more or less marked in proportion as the dilatation affects the left ventricle. Dyspnœa will be prominent in proportion as the right ventricle is the seat of dilatation. The recumbent position, with the head low, may be insupportable, and in an advanced stage, the suffering from defective hæmatosis may amount to orthopnœa. Occurring in paroxysms, this difficulty of respiration constitutes the affection called cardiac asthma. Exercise, and mental excite-

[1] Bellingham on Diseases of the Heart, Part 2. Dublin, 1857, p. 465.

ment exasperate the symptoms, particularly those referable to the respiration. More or less cough and expectoration are usually present. The abdominal viscera, as well as the lungs, are in a state of passive congestion. Owing to this state, the liver is often more or less enlarged permanently, and may be found to augment rapidly in size when, from any cause, the circulation is temporarily embarrassed in an unusual degree, resuming its former dimensions when the paroxysm ends and the heart recovers its habitual strength.[1] The digestive functions are weakened, but nutrition may be sufficiently active; patients do not always emaciate. The urine is not abundant, and may be found slightly albuminous, which is due to renal congestion and not necessarily indicative of structural disease of the kidneys. Granular degeneration, or Bright's disease, is, however, associated, in a certain proportion of cases, with dilatation as with hypertrophy. Finally, œdema occurs, first, manifested in the lower extremities, thence extending over the body, and effusion into the serous cavities succeeds, constituting general dropsy.

This is an enumeration of the more important of the symptoms belonging to cases of enlargement in which dilatation predominates, but it is to be borne in mind that, in general, valvular or other lesions co-exist, which, after inducing more or less hypertrophy in the great majority of cases, have at length led to the superinduction of dilatation; and, under these circumstances, it is difficult to say to what extent the symptoms distinctive of this stage of the disease may not be due to the causes of the dilatation, in other words, to the degree and duration of the concomitant lesions. It can hardly be doubted that considerable importance is to be attached to the dilatation in the production of the symptomatic phenomena which have been mentioned.

The pathological effects of dilatation are in a great measure embraced in the foregoing account of the symptoms. The dilatation is the result of weakness of the cardiac walls, together with an accumulation of blood within the cavities; and, on the other hand, it is the cause of further diminution of the power of the heart's action, and consequent over-repletion. It involves, therefore, an intrinsic tendency to increase. The evils incident to enlargement are mostly referable to dilatation. Little or no inconvenience is felt so long as the heart is hypertrophied, and the capacity of its

[1] Stokes on the Heart and Aorta.

cavities not increased. But in proportion as the latter takes place, the quantity of blood to be propelled from the cavities is greater, and the ability of the muscular walls to contract sufficiently for its propulsion is lessened; hence, inadequacy of the motive power of the central organ to carry on the circulation. This inadequacy increases in more than an arithmetical ratio as the dilatation progresses. The immediate effect on the vascular system is passive congestion, arising not alone from the defective propelling power of the heart, but from the obstacle presented to the return of blood to this organ by the accumulation within its cavities. The ulterior effects dependent on congestion are, embarrassment of the functions of the important organs of the body, serous transudation or dropsy, and, occasionally, hemorrhage. An occasional effect of great dilatation conjoined with extreme feebleness of the heart's action, is the formation of coagula within the cavities. There is reason to believe that in some instances in which the accumulation is excessive, and the contraction of the walls extremely feeble, the blood coagulates during life, and proves the immediate cause of a fatal termination. The formation of coagula in the heart during life will receive distinct consideration in a subsequent chapter.

Physical Signs and Diagnosis of Dilatation.

The physical signs of enlargement of the heart have been already fully considered. The signs distinctive of dilatation are now to be noticed. The several methods of exploration which furnish evidence of enlargement, supply certain indications pointing to dilatation in distinction from hypertrophy. The indications derived from percussion relate to the form of the area of deep dulness. If the boundaries of the heart are delineated on the chest by careful percussion, the transverse dimensions of the area preponderates over the vertical, in proportion as the dilatation predominates over hypertrophy. This corresponds to the difference as regards the form of the heart, which has been mentioned. The outline which the heart presents is wedge-shaped or nearly square if the dilatation be excessive. Palpation furnishes negative characters more readily available and striking. The sluggish, powerful apex-beat of hypertrophy is wanting; also the elevation of the ribs and the heaving of the præcordia. The impulse of the apex is feeble, and may be suppressed. The movements of the organ, owing to the

extended space in which it is in contact with the thoracic walls, are sometimes obscurely felt, and oftener visible in two, three, and even four intercostal spaces, which together present an appearance of fluctuation, or, as called by Dr. Walshe, quasi undulation. In some cases in which the thoracic walls are thin, and the intercostal spaces wide, the heart, as has been remarked, seems to be almost exposed to the vision and touch.[1] Auscultation furnishes certain distinctive points pertaining to the heart sounds. Both sounds are feeble in comparison with their augmented intensity in cases of hypertrophy, but the first sound is disproportionately weakened. The first sound is still more altered in character; it becomes short and valvular, resembling in these respects the second sound. The latter alteration, although distinctive of dilatation, as contrasted with hypertrophy, is not peculiar to the former, and its true explanation has not been understood. It is due to the absence of the element of impulsion in the first sound. This element is deficient or wanting whenever the left ventricle lacks the muscular power necessary for its production. In hypertrophy this element is exaggerated owing to the increased force of the ventricular contractions; and in dilatation it is feeble or absent owing to the feebleness which at the same time render the apex-beat weak or inappreciable. But this element is also impaired or eliminated when, from other causes than dilatation, the muscular power of the heart is weakened. The intensity of the first sound is diminished disproportionately to that of the second sound, and it is also short and valvular like the second sound, in cases of fatty degeneration, softening in typhus fever, and even of hypertrophy, when the power of the ventricular walls is greatly reduced. An adventitious sound or murmur is said to accompany the first or systolic sound in some instances of dilatation not complicated with valvular lesions. As a rule, a murmur is not present unless the latter coexist, or the blood have undergone those abnormal changes which occasion a murmur without any organic affection of the heart. This point will be noticed in treating of murmurs in connection with valvular lesions. Inspection shows in certain cases the quasi-undulatory movements within the præcordia which have been mentioned as also determinable by palpation. They are better perceived by the eye than by the touch. Inspection and mensuration may show an abnormal prominence of the præcordia. In the rare cases of dila-

[1] Racle, op. cit.

tation with attenuated walls, it may be true that enlargement of the præcordia never occurs. This is not true, however, of all the cases in which dilatation predominates over hypertrophy. Without discussing the question whether enlargement by dilatation as well as by hypertrophy may not give rise to præcordial projection, this result may be produced by the hypertrophy before the supervention of dilatation which subsequently becomes predominant. Absence of præcordial prominence does not then belong among the negative signs of enlargement by dilatation.

In the diagnosis of enlargement by dilatation, assuming the fact of enlargement to be ascertained, symptoms (as distinguished from signs) have considerable weight. Passive congestions, lividity, feeble pulse, and dropsical effusion, in fact, constitute evidence almost, if not quite, conclusive. The obstruction due to valvular lesions so generally associated with enlargement, it is true, contribute towards the production of these symptoms; but, as will be seen when valvular lesions are considered, the obstruction due to these rarely, if ever, give rise to the effects just mentioned until dilatation of the cavities of the heart has taken place. With the aid of the physical signs, the discrimination between predominant dilatation and predominant hypertrophy may generally be made with confidence. The cases in which there is room for doubt are those of hypertrophy when, from any cause, the muscular power of the heart is notably weakened. The differential diagnosis is of importance with reference to prognosis and treatment. The prospect of life and tolerable health is less in proportion as dilatation predominates, and the management involves attention to incidental events, which do not occur so long as hypertrophy preponderates. For the convenience of comparison with the physical signs distinctive of hypertrophy (see page 71), the signs distinctive of dilatation are embraced in the following summary.

SUMMARY OF THE PHYSICAL SIGNS DISTINCTIVE OF ENLARGEMENT BY DILATATION.

1. *Percussion.*—The transverse dimensions of the space occupied by the heart greatly exceeding the vertical, the form of this space corresponding to the wedge-like or square form of the organ when the dilatation is excessive.

2. *Palpation.*—The apex-beat devoid of abnormal force and in some instances suppressed. Absence of heaving movement of the ribs and præcordia.

3. *Auscultation.*—The element of impulsion of the first sound deficient or absent, and the sound short and valvular, in these respects resembling the second sound.

Treatment of Dilatation.

With certain qualifications, the indications for the treatment of dilatation are the same as in cases of predominant hypertrophy. The impediment to the circulation dependent on the lesions which coexist in the great majority of cases cannot be removed, but the effects may be mitigated by avoiding extrinsic causes which excite unduly the action of the heart. Bloodletting is called for much more rarely, and is to be employed with greater circumspection than when hypertrophy preponderates. Limiting the attention to the diminution of the mass of blood, it might seem that this measure would fulfil an important indication. But it is to be considered that bloodletting impoverishes the blood by its spoliative effects, and the secondary consequences are weakness and irritability of the muscular structure of the heart. These consequences are hurtful to an extent greatly overbalancing the advantage of temporarily diminishing the quantity of blood to be circulated. Before resorting to this therapeutical measure, the physician should be satisfied not only that the impediment is aggravated by an overplus of the mass of blood, but that the organized elements, viz., the corpuscles, which are disproportionately diminished by bloodletting, are not already deficient. No advantage to be derived from this measure can compensate for the evils of anæmia. Bearing in mind the immediate effects of bloodletting on the composition of the blood, and the secondary effects, due to impoverished blood, on the muscular structure, the cases in which it is called for seldom, if ever, occur. These remarks will, measurably, but not nearly to the same extent, apply to other methods of depletion, viz., saline purgatives and diuretics. Perhaps it may be said that in cases of dilatation the latter methods should be employed to the entire exclusion of bloodletting. Excessive muscular exercise, mental excitement, and other extrinsic causes exciting unduly the action of the heart, are to be avoided. Warmth of the external surface, and revulsive measures to attract blood to the extremities, are indicated oftener and more strongly in cases of dilatation than in cases of hypertrophy.

The measures which in hypertrophy are pursued in order to prevent dilatation, are not less indicated when the latter exists. The great end in the management is to increase the muscular power of the heart. For this end, the diet should be as highly nutritious as possible, and the quantity of liquid ingesta as small as is compatible with comfort. In the arrangement of diet, the state of the digestive organs is to be consulted. Imperfect or labored digestion involves excited action of the heart, and is to be carefully avoided. When indigestion exists, palliative remedies are to be prescribed; and remedies to improve the digestive function, viz., tonics and the judicious use of stimulants, constitute an important part of the treatment. Preparations of iron are especially indicated if there be anæmia. Constipation is to be prevented. Exercise, within certain limits, is to be enjoined. The injury arising from excessive muscular exertion has been referred to; but an extreme of quietude is not less hurtful. How is the judicious mean to be determined? The experience of the patient must be the guide. An amount or kind of exercise which excites unduly the action of the heart or occasions dyspnœa is to be abstained from; but exercise short of these effects will be useful. Patients who follow avocations which involve manual labor will, in general, do better to pursue their callings, observing the precaution just mentioned, than to relinquish all occupation. The necessity for an undue amount of labor in order to obtain a livelihood is a calamity for persons affected with cardiac disease; but a condition in life in which there is no other motive for exertion than the attainment of health is sometimes equally calamitous. Patients of the latter class should be encouraged to engage in sports which afford the requisite exercise, and, at the same time, interest the mind, such as shooting, fishing, and travelling. An advantage of no small account, incidental to pursuits which involve both exercise and mental occupation, accrues from the diversion of mind and cheerfulness which they promote. Depression and gloomy forebodings are to be obviated as far as possible, and with a view to this, as much encouragement should be given as the nature of the case will permit. In a large proportion of the cases which the physician meets with in practice, he may conscientiously encourage hopes, not of cure, but of tolerable health for an indefinite period. The common notion that disease of heart generally ends in sudden death may be removed by positive assurances of its falsity.

Remedies to allay undue excitement and irregularity of the

heart's action are indicated in cases of dilatation, as well as in cases of hypertrophy. The same remedies are indicated in both forms of enlargement; but they are to be employed with more caution in the former than in the latter. The danger of weakening or retarding too much the muscular action of the heart is far greater in cases of dilatation. Anodynes, digitalis, aconite, etc., are serviceable, but must not be pushed beyond the effect of tranquillizing the action of the heart, incurring risk of weakening the muscular power of the organ.

The paroxysms of dyspnœa or orthopnœa, sometimes the source of great distress in cases of dilatation, are to be palliated by antispasmodic remedies and revulsive applications. Of the former, the ethers, and of the latter, sinapisms, dry cupping, and stimulating pediluvia are the most efficient.

The treatment of dropsy dependent on cardiac disease is deferred till after the consideration of valvular lesions.

CHAPTER II.

LESIONS, EXCLUSIVE OF ENLARGEMENT, AFFECTING THE WALLS OF THE HEART.

Atrophy, with diminished bulk of heart—Fatty growth and degeneration—Symptoms and pathological effects of fatty growth and degeneration—Physical signs and diagnosis of fatty growth and degeneration—Treatment of fatty growth and degeneration—Softening of the heart in typhus and typhoid fever and other affections—Treatment of softening of the heart—Induration of the heart—Cardiac aneurism—Rupture of the heart—Carcinoma, tuberculosis, extravasation of blood and cysts.

Exclusive of enlargement, the heart is liable to various lesions affecting the walls of the organ, to some of which allusion has been already made, as standing in a causative relation to dilatation. Atrophy, with diminished bulk of the heart, is one of these; fatty growth and degeneration constitute others; other lesions are, softening and induration, and in this category may be included aneurism of the heart and rupture. This chapter will be devoted to the consideration of these different organic affections, taken up in the order in which they have just been mentioned.

ATROPHY WITH DIMINISHED BULK OF THE HEART.

The muscular substance of the heart is sometimes diminished, the cavities not being enlarged, but, on the contrary, their capacity lessened. The organ is reduced in size below the normal limits. In the adult subject it may resemble in bulk the heart of a child. The weight corresponds to the diminution in size. This reduction in size and weight does not involve necessarily any notable change in the appearance of the organ in other respects, the only obvious deviation from the normal condition being the diminution in volume and in the thickness of the ventricular walls.

This is undoubtedly to be considered as an organic affection of the heart, but it very rarely, if ever, occurs except in harmony, so to speak, with other morbid conditions, and under circumstances in which it neither occasions unpleasant consequences, nor claims attention in a therapeutical point of view. It is incidental to chronic diseases of long duration, characterized by gradual, progressive emaciation. It is observed in some cases of pulmonary tuberculosis, and more especially in cases of carcinoma. It is said to follow, in some instances, pericardial adhesions and calcification of the coronary arteries; but its dependence on these lesions does not appear to be established. It is observed, in some instances, in connection with a superabundance of fat on the exterior of the heart, and may be due, in these instances, as in cases of pericardial adhesions, to mechanical pressure of the organ continued for a long period. The conditions generally giving rise to it are diminution of the mass of blood, and of its nutritive materials—conditions involving diminished exertion of the muscular power of the organ. The heart wastes like other muscles when badly nourished and insufficiently exercised. But, under the circumstances, that is, in view of coexisting tuberculosis, or carcinoma, or some other affection, which, like these, terminates fatally after slowly progressive emaciation, the cardiac atrophy, so far from being an evil, may perhaps belong among the conservative provisions of which the pathological history of even the most fatal forms of disease furnishes illustrations.

The symptoms of atrophy of the heart, it is sufficiently clear, must be those which denote feebleness of the circulation; but inasmuch as an enfeebled circulation due to other morbid conditions, precedes and gives rise to the atrophy, it must be difficult to decide to what extent the symptoms are dependent on the latter. Nor are the symptoms denoting feebleness of the circulation distinctive of this particular lesion of the heart. They are incident alike to dilatation, fatty degeneration, softening, &c. The physical signs are much more distinctive, and, in fact, suffice for the diagnosis. The boundaries of the superficial and deep cardiac regions are within the extreme limits of health; the apex-beat is indistinct or wanting, and the heart-sounds are abnormally feeble, and may be inappreciable. In a patient under observation at the time I am writing, a clear, vesicular resonance on percussion is elicited over the entire præcordia. The respiratory murmur is quite intense and normal over the whole præcordial space, a fact which excludes

emphysema of the portion of lung overlapping the heart. There is in this case no superficial cardiac region; the anterior borders of the heart appear to meet. The left boundary of the deep cardiac region is sufficiently defined by the percussion-sound, and falls half an inch within the nipple. The apex-beat is not felt, and the heart-sounds are nowhere discoverable. There is evidently considerable atrophy in this case, yet there are no symptoms pointing to cardiac disease. The patient has for several years been affected with pulmonary tuberculosis, which is either non-progressive, or advancing very slowly.[1]

As already stated, atrophy of the heart does not call for medical treatment.

FATTY GROWTH AND DEGENERATION.

With the undue accumulation of fat are connected lesions quite different in character and importance, according to the difference of situation in which the fat accumulates. More or less fat is generally present in health on the outer surface of the heart after early infancy, especially on the right ventricle, at and near the base of the organ. It accumulates in this situation to an abnormal extent in some cases. A moderate amount of over-accumulation is frequently met with in post-mortem examinations, when there had been during life no symptoms of heart disease. If the quantity do not considerably exceed the normal average, although it must in some measure embarrass the movements of the organ, it does not occasion any serious results or appreciable inconvenience. When the accumulation is excessive, however, from its weight it leads to enfeebled muscular action and consequent weakness of the circulation. It may also favor dilatation if, from other causes, the blood accumulate unduly within the cavities of the heart. Without these concurrent causes, it may induce atrophy with diminished size of the muscular portion of the heart. This variety of fatty heart occurs after the middle period of life, in persons who present evidence of an "adipose diathesis,"[2] viz: accumulation of fat in

[1] Case of Thos. Carr, Hospital Records, vol. xiii. page 87.

[2] This term is borrowed from my friend, Prof. Gross. *Elements of Pathological Anatomy*, third edition, 1857. Dr. Bellingham also makes use of the term "fatty diathesis." *Treatise on Heart*, part ii., 1857.

different organs and beneath the integument, constituting corpulency. Not unfrequently, however, it occurs in persons who are not corpulent.[1] The heart is sometimes completely encased in a thick layer of adipose substance, which alters, in a marked degree, the external appearance and form of the organ. The volume of the heart is often increased not alone by the fatty deposit, but by more or less dilatation. Beneath the fatty layer the muscular substance may not present any structural change. It is, however, generally unusually pale, and the texture softened.

The extension of fatty growth between the muscular fibres is followed by more serious consequences than when the deposit is limited to the surface of the organ. The pressure upon the fibres induces greater functional weakness, and, at length, atrophy. The power of the heart in propelling the currents of blood and in resisting the force of distension from accumulation within the cavities is proportionately lessened. Hence, feebleness of the circulation and proneness to dilatation in proportion to the amount of deposit in this situation. The deposit in this situation may be in the form of adipose vesicles and infiltrated oily matter.

Another variety, much more serious, and differing essentially in character, is that commonly known as fatty degeneration. The fat is deposited in the form of oil-globules within the sarcolemma. It replaces the muscular substance and constitutes another form of fatty atrophy. This variety may be associated with the preceding varieties of fatty heart, but it occurs independently of the latter. It affects more especially the left ventricle, while the varieties consisting of abnormal growth of the adipose vesicles are most abundant on and within the right ventricles. It may be pretty uniformly diffused over the left ventricle or the whole heart, but it is oftener confined to circumscribed patches or strips. The portions affected assume a yellowish or fawn color, which is somewhat characteristic, and if the heart be affected in disseminated patches it presents a mottled aspect. Examined with the microscope, the striæ or transverse markings of the fibres are indistinct or wanting, and in place of the proper contents of the sarcolemma, it contains oil-globules in more or less abundance according to the amount of degeneration. It is evident that in proportion to the degree and extent of this

[1] Of 49 cases analyzed by Dr. T. K. Chambers (*Decennium Pathologicum*), it was associated with general corpulence in 20, and occurred in persons not corpulent in 29. *Vide* Bellingham, op. cit. part ii.

structural change, the muscular power of the heart must be irrecoverably weakened. It is proportionately incapacitated to propel the blood with adequate force, and more readily yields to distension from the accumulation of blood. The portions of the organ which have undergone fatty degeneration are soft and friable, and it will be seen presently that when rupture of the heart takes place, it is owing generally to this structural change having occurred. Cases are reported in which apparently the greater part of the muscular substance had disappeared, the fibres preserving their outline, but containing fat instead of their proper anatomical elements. The auricles may be the seat of this fatty change, but much more rarely than the ventricles. For a fuller description of the gross and microscopical appearances presented in fatty degeneration, the reader is referred to works on morbid anatomy.[1]

The distinct pathological character of fatty degeneration, as compared with fatty growth upon the heart and between the muscular fibres, is a point of importance. The latter is, in fact, hypertrophy of the adipose tissue, while the former involves more properly an abnormal deposit. The term *degeneration* implies a conversion of the muscular substance into fat. That the mechanism of fatty degeneration does involve this transformation is the view entertained by some distinguished pathologists.[2] The muscular substance, according to this view, undergoes a metamorphosis, the same elements recombining to form the fatty matter, as muscular tissue after death is supposed to be converted into adipocere. If this view be correct, it is not strictly accurate to call the fatty matter a deposit; it is not, at all events, deposited primarily as fat, but as the substance of the muscle. Nor is the change due to perverted nutrition; it is due to a chemical, not a vital process. A more philosophical explanation attributes the change to a process of replacement rather than conversion. The fat is strictly an abnormal deposit, which takes the place of the muscular substance. The change, agreeably to this explanation, does not consist, properly speaking, in a degeneration of structure, but in the substitution of one anatomical element for another, and it has been proposed to employ, as a more accurate mode of expression, the term *substi-*

[1] Rokitansky and Jones & Sieveking may be consulted for this object.

[2] For the evidence to be adduced in support of this doctrine, the reader is referred to an article by Dr. Richard Quain (in *Medico-Chirurgical Transactions*, vol. xxxiii.) on Fatty Disease of the Heart.

tution instead of degeneration.[1] They who adopt the latter view regard atrophy of the muscular tissue as the first step in the local pathological process. The anatomical elements within the sarcolemma disappear by absorption, and fat is deposited in their place. It is, perhaps, as reasonable to suppose that the primary change is the fatty deposit, the removal of the anatomical elements of the muscular tissue taking place subsequently, the atrophy thus being not a prior, but a consecutive condition.

On what antecedent morbid conditions are the different forms of fatty disease of heart dependent? Morbid growth or hypertrophy of the adipose tissue, as already stated, is often associated with that tendency to superabundance of fat which constitutes obesity. This tendency is directed towards the heart, after middle life, in persons of indolent and luxurious habits, and who are addicted to the use of alcoholic beverages. Active exercise and a well-regulated diet serve to protect the heart against accumulation of fat, even when the adipose diathesis is marked. Fatty degeneration occurs independently of this diathesis. The conditions on which it depends are not well ascertained. It is a question whether it involves a prior alteration of the blood, or is to be regarded as an effect of conditions purely local. The latter view is favored by the doctrine that muscular atrophy precedes the fatty deposit; and, on the other hand, if the deposit takes precedence, a predisposing condition of the blood is reasonably inferred. The latter supposition is sustained by the fact that, although this variety of fatty heart occurs in persons who are not corpulent, it is usually found in association with fatty degeneration in other parts, especially the liver, spleen, and the arterial coats. This fact points to a blood-crasis or diathesis. Prof. R. W. Smith, of Dublin, has reported cases in which free oil was collected, after death, in considerable quantity, from the blood contained in the vessels and heart-cavities.[2] As suggested by Robin, the oil in these cases may have exuded from the tissues as a result of *post-mortem* decomposition, and in one of the cases its presence in the cavities of the heart may be explained by the fact that rupture of the ventricle had occurred. Dr. Quain, who regards the change as a conversion of elements, attributes the affection to impaired nutrition, frequently dependent on coexisting obstruction of

[1] Ch. Robin, *vide* Chimie Anatomique; also Dictionnaire de Médecine (Nysten), 1855, art. Dégénération.

[2] These cases are reported in the *Dublin Journal of Medical Science*, first series, vol. ix. p. 411. See Stokes on *Diseases of the Heart and Aorta*.

the coronary arteries. Of eighty-three cases analyzed by him, these arteries were ossified or obstructed in twenty-five. Rokitansky, having observed it often in connection with pericarditis and endocarditis, thinks it may occur as a sequel of these affections. It is observed in cases of enlargement. Of forty-nine cases analyzed by Dr. T. K. Chambers, the heart was enlarged in twenty-nine. In twenty-three of these cases dilatation predominated. Its occurrence with other causes doubtless contributes to dilatation; but, in view of the infrequency of the latter, except in connection with valvular or other affections impeding the circulation, it cannot often be referred exclusively to fatty degeneration. On the other hand, it is not improbable that enlargement of the heart may lead to fatty degeneration.[1] This opinion is held by Rokitansky.

Symptoms and Pathological Effects of Fatty Growth and Deposit.

Although the different forms of fatty disease differ pathologically, their general effects are similar. They induce alike weakness of the heart, and symptomatic phenomena due to enfeebled circulation. But they by no means induce these effects in an equal degree. A considerable accumulation of fat upon the heart, as stated already, may exist without giving rise to symptoms which point to cardiac disease. The effects are more marked if the fatty substance penetrate between the muscular fibres, or if the organ become infiltrated with fatty matter. They are still more strongly marked in cases of oily degeneration in which atrophy of the muscular tissue has occurred to much extent. Each variety, of course, is important, or serious, in proportion to its diffusion and amount; but microscopical observers have found considerable degeneration in hearts in which disease had not been suspected either from the symptoms during life or the general appearance after death. The

[1] Mr. Grant, of London, has lately investigated the pathological conditions in cattle fattened for exhibition, and finds that fatty degeneration of the muscular tissue, and especially of the heart, is a uniform result of the system of overfeeding pursued. This is of importance with reference to the connection of fatty degeneration and dietetic habits in the human species. *Vide* "Evil Results of Overfeeding Cattle. A new inquiry, fully illustrated by colored engravings of the heart, lungs, etc., of the diseased prize cattle lately exhibited by the Smithfield Cattle Club, 1857. By Frederick James Grant, M. R. C. S., etc. London, 1858." (*Brit. and For. Med.-Chir. Rev.*, July, 1858.)

great extent and degree of degeneration found in some instances are remarkable.

The symptomatic phenomena referable directly to the heart and circulation (exclusive of physical signs) are not distinctive. The pulse, if the heart be not very greatly weakened, may be natural in frequency and perfectly regular. It will lack force; but the differences in this respect are so great among healthy persons, owing to a variety of circumstances, that this quality of the pulse does not possess much significance. Notable slowness of the pulse has been observed, the number of pulses per minute falling to twenty or thirty, and even much lower in some cases. They have been observed as low as eight and nine per minute.[1] In an advanced stage of disease, intermittency, irregularity, and great frequency, conjoined with extreme feebleness, are liable to occur. These characters, however, belong equally to the pulse in cases of dilatation; they do not indicate specially, of themselves, fatty disease. The pulse and systemic circulation will, of course, furnish manifestations of cardiac weakness more marked in proportion as the disease affects the left ventricle. General dropsy does not occur as a result of merely weakness of the heart, exclusive of valvular lesions and dilatation. Sense of oppression at the præcordia, palpitation, and a tendency to syncope, are other symptoms referable directly to the heart and circulation. I have also observed a notable degree of capillary congestion of the extremities.

Dyspnœa on exercise is present in proportion as the right ventricle is weakened. But this symptom does not represent exclusively weakness of this ventricle. If the contractions of the left ventricle are feeble and incomplete, the left auricle necessarily becomes distended, and pulmonary congestion ensues. Hence more or less dyspnœa is present when fatty degeneration is confined to the left ventricle, provided its muscular power be considerably compromised.

These and other symptoms which might be mentioned are common to all affections compromising, beyond certain limits, the power of the heart's action. They point only to some cardiac trouble involving diminished power of the organ. Considered alone, they are not diagnostic of the affections under consideration.

[1] *Vide* memoir on "Slow Pulse," by Mr. Richardson, *Dublin Quarterly Journal*, vol. xiv. Dr. Bellingham remarks, with reason, that this abnormal slowness involves an abnormal cerebral condition superadded to the heart-affection.

Certain events pertaining to the nervous and respiratory systems have been supposed to be highly significant of fatty degeneration of the heart. The occurrence of seizures resembling apoplexy, appears to have been first observed by Dr. Cheyne.[1] Subsequently cases have been reported by Drs. Adams, Law, and Stokes, of Dublin. These attacks are characterized by the sudden loss of consciousness, and recovery without paralysis. In some cases they recurred frequently, in the end proving fatal, and on dissection the brain presented no morbid appearances adequate to explain their occurrence. The heart had undergone extensive fatty change, and the cavities were dilated.[2] An instance, probably illustrating the association of this pseudo-apoplectic affection and fatty degeneration, the existence of the latter being determined by clinical evidence, came under my observation in 1856, in the case of a hospital patient, aged 56, admitted in a state of unconsciousness. His eyes were open, his expression vacant, and he made no effort to reply to questions. The skin was cool; the pulse small and not accelerated. The persons who brought him to the hospital could give no account of the history, except that he was found in his present condition. He made no effort to assist himself on his way to the hospital. Respirations were normal. Brandy and nourishment alone were prescribed. The next morning he manifested some consciousness, but appeared idiotic; smiled when questions were put to him, and said nothing. The following day the improvement was more marked. Said he was quite well. Could give no account of his attack. He speedily convalesced, and afterwards replied to questions intelligently. He declared that he had no recollection of his attack, nor of his being brought to the hospital. The *arcus senilis* in this patient was well marked, more so in the right than in the left eye. The pulse was 80, and quite feeble; capillary congestion marked on the hands, and the face pallid. The heart-sounds were extremely feeble. No impulse seen or felt, except a very slight movement in the epigastrium. No murmur. He was accustomed to drink about half a pint of whiskey daily. He left the hospital after a fortnight, and the subsequent history is unknown.

[1] Dublin Hospital Reports, vol. ii. Case is reported in Stokes on Diseases of the Heart and Aorta, Am. ed., p. 319.

[2] This statement with respect to dilatation applies to the cases given by Dr. Stokes, op. cit. The reader will find the subject treated at some length in that work.

The nature of the pathological relation existing between attacks of pseudo-apoplexy and the cardiac affections under consideration, with our present knowledge, can only be conjectured. That it consists in disordered cerebral circulation due to the weakened condition of the heart, is not probable, inasmuch as the muscular power of the organ is equally reduced in cases of simple and complicated dilatation. It is more rational to suppose that some intermediate morbid conditions are involved; conditions not dependent on cardiac disease, but associated, more or less frequently, with it. In one of the cases reported by Dr. Stokes, calcification of the cerebral arteries existed in a notable degree. In fact, further clinical observation is necessary to establish the existence of a fixed pathological connection between these attacks and fatty degeneration, or any disease of the heart. I have met with an instance in which attacks analogous to those described by the author first named, occurred from time to time for several years, when no other evidence of disease of the heart existed. A gentleman, aged 33, of a highly nervous temperament, as often as once a month, without any apparent exciting cause, has what are called "fainting fits," during which, for a few moments, he loses his consciousness. The extremities are cold, and sometimes the attacks are accompanied by slight rigor. Febrile reaction does not follow. There are no convulsive movements, nor disturbance of the respiration. The patient consulted me under an impression that there existed disease of the heart, but the physical signs denoted in all respects a normal condition of this organ. In addition to a life of ease and indolence, this gentleman had for years been accustomed to the daily indulgence of the venereal act.[1]

A peculiar aberration of the respiratory movements, first described by Dr. Cheyne, but not considered by him as significant of any special cardiac lesion, is regarded by Dr. Stokes as characteristic of great weakness of the heart incident to fatty degeneration. This symptom is thus described by the distinguished author just named: "It consists in the occurrence of a series of inspirations, increasing to a maximum, and then declining in force and length, until a state of apparent apnœa is established. In this condition the patient may remain for such a length of time as to make his attendants believe that he is dead, when a low inspiration, followed by one more decided, marks the commencement of a new ascending

[1] Case of ———, Private Med. Records, vol. x. p. 253.

and then descending series of inspirations. This symptom, as occurring in its highest degree, I have only seen during a few weeks previous to the death of the patient."[1]

Fatty degeneration of the iris, giving rise to the appearance well known as the *arcus senilis*, has been observed in cases of fatty degeneration of the heart. It has been supposed to be valuable as a symptom from its frequent coincidence with the latter. Its precise value is yet to be determined by clinical experience. It may be regarded as at least denoting a condition of the system favorable for fatty degeneration, and, taken in connection with the symptoms and signs relating directly to the heart, it is not without diagnostic import. The degree of significance which belongs to it depends, of course, on the frequency or constancy of the association. With our present knowledge it is certainly insufficient as a basis of the diagnosis of fatty degeneration of the heart, exclusive of local signs and symptoms; and, on the other hand, its absence is not to be considered as proof that the cardiac affection does not exist.[2]

According to Dr. Walshe, failure of the sexual inclination and power is to be included among the pathological effects. He states that in one of the best marked cases of the disease which he ever saw, clinically speaking, in a person under forty-five years of age, he was consulted for impotency, without reference to the cardiac symptoms.

Paroxysms of pain and dyspnœa, constituting *angina pectoris*, may occur in conjunction with fatty degeneration. No special pathological relation, however, exists between these affections inasmuch as angina pectoris is associated with other organic lesions of the heart. Being common to different cardiac affections, I shall treat of it under the head of functional disorders of the heart.

The pathological effects of fatty growth and degeneration are serious in proportion as they involve structural change, or, in other words, atrophy and impaired consistence of the walls of the heart.

[1] On the Diseases of the Heart and Aorta, Am. ed., p. 340.

[2] Mr. Edward Canton, of London, first ascertained that the *arcus senilis* is due to fatty degeneration of the cornea, and advocated the opinion that, when present, under the age of 40, it is invariably associated with fatty heart, vide *London Lancet*, 1850 and 1851. As regards the frequency of its association with fatty heart, clinical experience is thus far discrepant. Vide *Williams' Principles of Medicine*, and article by Dr. Hopkins, in *Am. Journ. of Med. Sci.*, Jan. 1853. Also an Essay "On the Symptomatic Value of the Arcus Senilis; with a tabulated statement of Seventy-two Cases." By Benjamin Lee, A. M., M. D., in the *Am. Med. Month.*, Sept. 1856.

Hence, fatty degeneration is the form of disease which is especially attended with notable disorder and danger. The atrophy in this affection differs from that previously considered under the head of atrophy, with diminished bulk of the heart. The muscular substance lost is replaced by the deposit of fat, which makes good the volume of the heart, but, of course, without supplying, in any measure, the loss as regards the function of the organ. Cases, however, of death simply from fatty degeneration of the heart must be exceedingly rare. The best anatomical specimens of the disease are obtained from bedridden patients of an advanced age. It may prove fatal, by leading to rupture. Sudden death, in some cases, also occurs in an attack of syncope; the cavities becoming overloaded, and the walls too feeble to propel the blood, the movements of the organ are suddenly arrested. Exclusive of these instances, which are extremely infrequent, a fatal result is generally not due directly to the cardiac affection, but to the concurrent effects of associated or superinduced pathological conditions. This, however, by no means renders the existence of fatty degeneration unimportant, either as regards diagnosis or treatment. Dr. Stokes has suggested that the presence of an amount of structural change, not sufficient to give rise to well-marked symptoms of cardiac disease, may serve to explain the disproportionate feebleness of the circulation, tendency to syncope, and the intolerance of bloodletting and other debilitating measures, which are sometimes observed in different affections. The probability of the coincidence of the affection under consideration, is to be taken into account in cases in which the fact cannot be positively determined.

Physical Signs and Diagnosis of Fatty Growth and Degeneration.

The accumulation of fat is very rarely sufficient to increase the size of the heart much beyond the limit of the variations in health. If, therefore, percussion show a considerable amount of enlargement, dilatation is to be inferred. Dilatation and fatty disease are not unfrequently combined, and the question at once arises, is the diagnosis of the latter, under these circumstances, practicable? The extent of enlargement of the heart can generally be determined with precision. By means of percussion and palpation, the space which the organ occupies can be delineated and measured. Now, if the enlargement be sufficient in extent to correspond with the asso-

ciated signs and symptoms, evidence of fatty disease is wanting; but if, on the contrary, the signs and symptoms denote a degree of cardiac weakness out of proportion to the enlargement, fatty disease may be strongly suspected; and, if other circumstances are present pointing to the latter disease, the diagnosis may frequently be made with much positiveness. The exclusion of valvular lesions is an important point in the diagnosis. If valvular lesions are not present, the coexistence of fatty degeneration is rendered highly probable by the fact of dilatation, the latter probably occurring in consequence of the former. It is to be borne in mind that the presence of valvular lesions by no means precludes the existence of fatty disease; but in cases in which valvular lesions, enlargement and fatty disease are combined, the diagnosis of the latter cannot certainly be made with positiveness. It may, however, be reasonably suspected when, under these circumstances, the weakness of the heart is greater than would be expected from the amount of dilatation.

Limiting the attention to cases in which fatty disease is the sole or paramount lesion, and in which the atrophic changes are sufficient to give rise to well-marked manifestations of a cardiac affection, what are the physical signs furnished by the different methods of exploration? Percussion shows moderate or no increase of the volume of the heart. This is an important negative point. The apex-beat, if felt, will be but little, if at all removed from its normal situation. The beat, if felt, is abnormally feeble, and it will be inappreciable if the heart be greatly weakened. Impulses elsewhere than over the apex, will, in general, not be discoverable. Inspection may disclose a very feeble movement over the apex, or none whatever. The diminished force or suppression of the apex-beat will depend, of course, on the extent to which the left ventricle is affected. The sounds of the heart are weakened. The first sound, more than the second, shows abnormal weakness. It is also short and valvular, resembling, in these respects, the second sound. The greater weakness of the first sound and its altered quality, are due to the effect upon the element of impulsion. This element is impaired more than the valvular element, and may be suppressed while the latter remains. The first sound may be wanting, the second sound being still heard; and, finally, both sounds may be extinct. The latter obtains in cases of a very great degree of fatty degeneration.

When in connection with these physical signs, there are present

symptoms denoting a cardiac affection, viz: feebleness, and perhaps irregularity of the pulse; palpitation and præcordial distress; dyspnœa on exercise, tendency to syncope, etc., there can be but little room for doubt that the heart is affected with fatty degeneration, especially if the patient have passed the middle period of life, if his habits of life have been luxurious and indolent, if he have been addicted to alcoholic beverages, if he have the *arcus senilis*, or if there be a tendency to obesity. The diagnosis is not difficult under these circumstances. It is less easy when the problem is to decide whether fatty degeneration exists in addition to enlargement and valvular disease; and also when the amount of degeneration is not sufficiently great to give rise to well-marked symptoms and signs of cardiac disease. The probability of the coexistence of this lesion with other affections which the physician is called upon to treat, is important to be taken into account in the interpretation of symptomatic phenomena, and the employment of therapeutical measures.

Treatment of Fatty Growth and Degeneration.

The general objects of medical treatment in cases of fatty growth and degeneration are threefold, viz., 1. To obviate and relieve the immediate effects of weakness of the heart; 2. To increase permanently the muscular power of the organ; and 3. To arrest or limit the accumulation of fat.

Of the immediate effects of the cardiac weakness incident to these affections, the more prominent are palpitation and præcordial distress, syncope, dyspnœa, and, possibly, apoplectiform coma. These effects occur generally in paroxysms, induced by causes which either temporarily increase the habitual weakness, or which, like exercise, mental excitement, etc., overtask the power of the heart. Some of the effects, however, may be more or less constant. The means of obviating and relieving them consist of measures to augment the force of the ventricular contractions; in other words, the use of remedies which act as cardiac stimulants. These are wine or spirits, ether, and the carbonate of ammonia. They are to be given more or less freely according to the urgency of the symptoms, that is, in proportion to the degree of cardiac weakness; and they are to be continued or repeated according to the persistence or recurrence of the paroxysms. Their habitual use is indicated if the effects are constant. The particular stimulants to be

selected must vary with reference to the habits of patients and the results of experience in individual cases. As regards quantity, they are to be graduated by the symptoms and by the relief afforded. It is impossible to formularize the means of fulfilling this object of treatment. Here, as well as with reference to the other objects of treatment, with a clear idea of the ends and means, the judicious practitioner will not be at a loss as regards therapeutical details. Without a proper knowledge of the pathological character of the immediate effects, serious errors of practice may be committed. Depletion by bloodletting or otherwise, and all measures tending to enfeeble still more the circulation, can hardly fail to be pernicious. In the attacks of pseudo-apoplexy, which have been referred to, whether immediately dependent on the heart or not, stimulants are not to be withheld on the supposition that the brain is congested. Cerebral congestion, it is to be borne in mind, may proceed from enfeebled power of the heart. In employing diffusible or alcoholic stimulants, the aim is not to excite the heart, but to strengthen its action. If they produce greater frequency of the pulse, the end is not attained. Their effect should be augmented force and volume of the pulse; and with this effect the frequency may be diminished and the rhythm become more regular. Remedies to retard the frequency of the pulse, such as digitalis, are of doubtful utility.[1] Revulsive measures, such as stimulating pediluvia, are useful, as in dilatation, by diverting the blood from the heart, and thus diminishing, for the time, the labor of the circulation. These remarks apply to remedial measures. It is hardly necessary to say that all the causes which either induce temporarily the habitual weakness or overtask the power of the heart are, as far as practicable, to be avoided and removed. Undue fatigue or depressing agencies of all kinds, physical and mental, violent muscular exertions, excitement, etc., belong in the category of exciting causes.

The second object, viz., to increase permanently the muscular

[1] M. Beau, of Paris, contends that digitalis not only diminished the frequency of the heart's contractions, but that, by a special action, it imparts to them increased strength and renders them more complete. To quote his language: "*La digitale est un tonique spécial du cœur; c'est, a vrai dire, le quinquina du cœur.*" (Traité Expérimental et Clinique d'Auscultation, etc., 1856, p. 372.) He states that this opinion was held by Saunders. If the opinion be correct, so far from being contra-indicated, the remedy is peculiarly adapted to the treatment of the affections under consideration, as well as cases of dilatation. I am not prepared to express either assent or denial, but there seems to me some foundation for the opinion.

power of the heart, is to be effected by tonic remedies, by an appropriate system of diet and regimen, and by judiciously regulated exercise. Tonic remedies are called for with a view to improvement of the appetite and digestion, if impaired or disordered. Quinia, bitter infusions, and the mineral acids, are often useful with reference to this end. The preparations of iron are especially indicated if anæmia exist. An anæmic condition is to be dreaded not less in these than in other affections of the heart; in all, the symptoms are greatly aggravated whenever it coexists. Flatulency and constipation are to be relieved by suitable remedies. With reference to the proper performance of the digestive functions, wine, spirits, or beer, in moderate quantity, may generally be taken with advantage. The dietetic course is of very great importance. The end is to contribute towards the healthy nutrition of the affected organ by rendering the blood rich in nutritive materials. For this end, the articles of diet should be highly nutritious and easily assimilated. The diet should consist of as large a proportion of animal food as the digestive powers will permit. The quantity of fluids should be restricted, in order that, while the blood is enriched in quality, the vessels and heart cavities shall not be unduly repleted, the labor of carrying on the circulation being proportionate to the mass of liquid to be circulated. Warm clothing is of importance to secure the distribution of a proper proportion of blood to the surface and extremities. Excesses of all kinds, in eating, drinking, venery, mental occupation or excitement, late hours, etc., are to be rigidly interdicted. Regular habits of life in all respects are important. Judiciously regulated exercise in the open air constitutes a part of the management not least in importance. Physical indolence predisposes to these affections; and, on the other hand, by habitual, systematic exercise, the heart is directly strengthened. Great caution, however, is to be observed in this part of the management. While judiciously regulated exercise is of great importance, injudicious excess may do much harm. The practical rule to be observed here is the same as in cases of dilatation. The patient is to be encouraged to take such exercise as he is able without experiencing inconvenience from dyspnœa or palpitation; in other words, undue excitation of the action of the heart is to be the limit. As the ability to endure greater and more prolonged exercise augments, the limit may be enlarged and extended. Violent exertions are never appropriate; walking, riding, rowing, the use of dumb-bells, etc., should be the

modes of exercise resorted to. Pedestrian and equestrian excursions, involving the mental interest of travelling, and hunting, and fishing, if pursued with zest, are especially to be recommended. Proper instruction and cautions are to be enjoined, as the tendency with many persons is, when once a system of exercise is undertaken, to push it to an extreme. The reader will have observed that thus far the principles of management do not differ materially from those to be pursued in cases of dilatation of the heart. The ends of management in dilatation and in fatty degeneration are, in fact, the same. The two lesions are often combined, and in treating the latter, an incidental object is to endeavor to prevent or limit the former.

The third object, viz: to arrest or limit the fatty accumulation, is peculiar to the affections under consideration. Theoretically considered, this object more clearly relates to fatty growth than to fatty degeneration. The former, in general, involves a tendency to the accumulation of fat in different parts of the body, or the adipose diathesis. The latter does not so generally involve this tendency or diathesis. That it does so to a greater or less extent is, however, probable. In fact, fatty growth and degeneration are often associated. The constitutional disposition, or, to speak more properly, the state of the blood favorable to obesity, may be controlled in a great degree by a diet adapted to this end, conjoined with habits of exercise. Fatty and saccharine substances should be interdicted, and articles abounding in amylaceous principles are to be sparingly allowed. The diet should consist of meat, bread, non-farinaceous vegetables, and certain kinds of fruit. Vegetables which are highly farinaceous, such as potatoes and rice, should be taken sparingly. By following this plan with perseverance, I have known an excessive corpulence greatly reduced, and the general health much improved. Caution is here necessary, lest the dietetic course be pushed to an extreme. The powers of the system are by no means to be lowered. A proper variety of alimentary principles is to be provided. It is only necessary that fatty substances be interdicted, and that those principles readily transformed into fat, viz: sugar and starch, constitute, relatively, a small proportion of the articles of diet.[1] Habits of exercise, it is well known, tend to prevent the

[1] A late writer, Dr. Markham, says: "I believe I have seen cod-liver oil, combined with steel, followed by very satisfactory results in supposed cases of fatty degeneration of the heart." (*Diseases of the Heart*, London, 1856, p. 289.)

It should be clear that the results were due to the oil, and not to the steel and

accumulation of fat. In this point of view, they are useful, in addition to their more direct effect, in increasing permanently the muscular power of the organ. Dr. Stokes thinks that exercise during summer or in a warm climate, when it is attended by copious perspiration, is especially useful, fatty principles being eliminated in considerable quantity from the surface.

As regards the success of treatment, so far as atrophy or degeneration of structure has taken place, the lesion must be considered as incurable. It is not probable that the substance which has disappeared is reproduced. But the heart, like the other important organs of the body, may sustain a certain amount of damage, while there still remain sufficient healthy tissue and functional power for life and health; and although that which is actually lost in structure cannot be recovered, the deficiency may be made up by increasing the development and vigorous action of the normal tissue which remains. Thus, a heart more or less unsound from fatty degeneration, may perhaps be rendered more efficient than it was even before the degeneration commenced. This statement is, of course, not applicable when the unsoundness is considerable. But assuming that a certain degree of weakness is inevitable for the remainder of life, if the progress of the structural affection can be stayed, and the capabilities of the organ developed and maintained, the condition of the patient may not be serious even with a considerable amount of unsoundness. With this view of the subject, the importance of an early diagnosis is sufficiently obvious.

The importance of recognizing fatty disease of heart in connection with inflammatory diseases affecting other organs, is not to be lost sight of. If there be grounds for suspecting the coexistence of fatty growth and more especially fatty degeneration, depletion and debilitating measures are to be employed with great circumspection. The question as to the presence of these affections will, in fact, oftener arise in such a connection, than in cases in which attention is called to a cardiac disease exclusively.

In conclusion, the subject of fatty degeneration of the heart offers a field for farther anatomical researches, especially by means of the microscope, and also for clinical observation, with a view to the development of facts which may be expected to shed light on its pathology, diagnosis and treatment.

other measures; and, also, that the disease was fatty degeneration, in order to base thereon the propriety of this remedy.

SOFTENING OF THE HEART.

Softening of the heart has been already considered as incidental to fatty degeneration. It will be noticed in a future chapter as a result of inflammation. Exclusive of these pathological connections, it belongs among the anatomical changes which are liable to take place in the course of the essential fevers, especially typhoid fever and typhus. Its occurrence in typhoid fever was observed by Laennec; but it was more fully studied by Louis, in those researches which established the natural history of that disease. In a certain proportion of cases of typhoid fever ending fatally, Louis found the muscular walls of the heart more or less softened. Sometimes the softening was limited to the left ventricle and sometimes it extended over both ventricles. When the diminished consistence was marked, the walls were notably relaxed and friable. The structure was easily torn and penetrated with the finger; the organ was flaccid, collapsing by its own weight, and not preserving its natural form, but retaining, like a wet cloth, any shape in which it was placed. When incised, the cut surfaces were dry and unpolished, and the color of the muscular tissue was purplish or livid. These alterations were not accompanied by any marked change in volume, nor by any of the products of inflammation.[1]

Softening of the heart, as occurring both in typhoid and typhus fever, has also been studied with much care by Dr. Stokes, his observations agreeing in all important particulars with those of Louis. Dr. Stokes has observed instances in which the external muscular layer of the left ventricle appeared to be converted into a homogeneous substance, all traces of muscular fibre being lost. He has also remarked an adhesive gummy liquid, with which the affected muscular substance was infiltrated.[2]

The softening, under these circumstances, is evidently due to a process which may be called acute, for it occurs early in the disease, and is most frequent and marked in the bodies of those who have died after a short career of the fever. Examinations made soon

[1] *Vide* Anatomical, Pathological, and Therapeutic Researches upon the Disease known under the Name of Gastro-enterite, etc. Translated by H. J. Bowditch M. D., 1836, vol. 1.

[2] Stokes on Diseases of the Heart and Aorta. Am. ed., p. 388.

after death, show that it is not due to cadaveric decomposition with which, without proper care, it is liable to be confounded. It may be associated with softening of other viscera—especially of the liver and spleen—but the voluntary muscles are not found to have undergone a similar change.

Of its pathological character and the mechanism of its production, all that can be said, with our present knowledge, is, that it is one of the secondary lesions dependent on the unknown morbid conditions which constitute the essential fevers. Whether the process involves, primarily, molecular changes due either to a morbid state of the blood, or to abnormal innervation; or whether the first step is the infiltration of a liquid which acts on the muscular tissue, remains to be ascertained. At present, the points important to be borne in mind with reference to their practical bearings are, its dependence on the essential pathological conditions in fever, whatever they may be, and its non-inflammatory character. That it produces, in certain cases, a degree of weakness of the heart, which renders the occurrence of the lesion an important event in the progress of the disease, contributing not unfrequently, to a fatal result, is not to be doubted. Clinical observation, however, furnishes evidence that it is a lesion which often occurs without leading to serious consequences, and that it admits of rapid and complete restoration. There is no ground for the supposition that it lays the foundation for permanent organic disease of the heart.

Softening of the heart, analogous to that just described, is not peculiar to the typhus and typhoid fevers. It may occur in other essential fevers. It has also been observed in purulent infection of the blood, in scorbutus, purpura, and other affections. As an element in the history of other diseases than the continued fevers, it has been less studied. Except as a secondary lesion developed during the progress of some general disease, it probably rarely, if ever, occurs independently of fatty degeneration or inflammation. But with reference to this point, as well as to the frequency of its occurrence in various diseases, further researches, with the aid of microscopical observation, are desirable. The microscope has been of great service in determining the dependence of softening on fatty degeneration in a large proportion of the instances in which it is observed after death.

The symptoms and pathological effects of softening of the heart are essentially the same as in fatty degeneration. They proceed from weakness of the organ, and are commensurate, as regards

their intensity, with the loss of muscular power incident to the lesions. There is this vast difference, however, in the two forms of disease, viz., the weakness due to fatty degeneration is permanent, while that incident to softening in fever is temporary, the lesion in the one case being incurable, and in the other case recovery taking place with certainty and rapidity, provided the fever end in convalescence. The pulse, in both cases, represents but imperfectly the degree of cardiac weakness. Other circumstances belonging to the circulation than the systole of the left ventricle affect materially the qualities of the pulse, especially the amount of resistance offered to the passage of blood through the capillary vessels from failure of the forces presiding over the circulation in these vessels, irrespective of the *vis à tergo* derived from the heart. On the other hand, feebleness of the pulse may proceed from other causes than softening of the heart, and, therefore, when strongly marked, it is by no means distinctive of this lesion. In cases of fever, moreover, the symptoms and pathological effects of cardiac softening are so intermingled with the phenomena pertaining to the febrile disease that it is impossible to isolate them. Functional weakness of the heart, without softening, is sufficiently common in typhus and typhoid fever, and the feebleness of the circulation, particularly as denoted by the pulse, may be as great in the one case as in the other. The diagnosis of softening, in short, cannot always be made with positiveness. Still, with the aid of physical signs and attention to certain points, the occurrence of this lesion may be determined, in some cases, with considerable confidence. It suffices for all practical purposes to consider the physical signs and diagnosis of softening as occurring in typhus and typhoid fever. The same considerations are applicable to the lesion when it takes place in other pathological connections in which it has not, as yet, been so fully investigated.

The physical signs of softening of the heart in fever were first thoroughly studied and their importance enforced by Dr. Stokes.[1] The signs are essentially those which belong to fatty degeneration, but, at the time of the original observations by Dr. Stokes, the latter affection was very imperfectly understood. To recapitulate these signs in the present connection, the apex-impulse becomes

[1] Dr. Stokes's original observations were made in 1837 and 1838, and published shortly afterwards. But the reader is referred to his late work on *Diseases of the Heart and Aorta* for a full consideration of the subject.

notably feeble or is suppressed; the intensity of both sounds is diminished, but the first sound is relatively much more weakened than the second. The first sound is altered in quality and duration, becoming short and valvular, in these respects resembling the second sound; in other words, it loses those characters which belong to the element of impulsion, and is analogous to the sound of the fœtal heart. These abnormal changes may be more or less strongly marked. The first sound is sometimes extinguished, while the second sound continues to be heard, and in some instances both sounds are inappreciable. The latter indicates great debility of the heart, and is very rarely observed. Taking place in the second week of the career of typhus or typhoid fever, when, during the early period of the disease, the impulse and sounds had been sufficiently intense, and had presented their normal characters, these signs denote either softening or simply functional weakness of the heart. Therapeutically, the indications are the same whether they are due to softening or to functional weakness, and, in a practical point of view, it is not of great importance to make the discrimination. As a matter of scientific interest, however, the differential diagnosis is deserving of attention. What, then, are the points which indicate softening? The researches of Louis show that softening is apt to occur rather early in the febrile career, at or soon after the end of the first week. Functional weakness, sufficient to give rise to the abnormal modifications of the sounds and impulse which have been described, is not likely to occur until a later period. If, therefore, the signs are present early, the presumption is in favor of softening. Functional weakness will be likely to be associated with marked general debility of the muscular system. It occurs in cases characterized by adynamia. Softening, being a special lesion, may take place when the voluntary muscles do not manifest extreme prostration. Want of correspondence, therefore, between the evidences of cardiac weakness and the condition of the general muscular system points to softening. Dr. Stokes attaches significance to the slow development of softening. The signs of the cardiac weakness, due to this lesion, are observed to become progressively but gradually marked, and then for some time steadily persist, while functional debility is liable to be rapidly induced, to vary from day to day, and is often less persistent. A point more distinctive than any other, which is available in a certain proportion of cases, relates to the results of a comparison of the heart-sounds in different situations within the præcordia. It

has been already stated that the softening is limited to or especially marked in the left ventricle. Now, as stated by Dr. Stokes, under these circumstances, the first sound may be louder at the inferior border of the heart, where it is derived from the action of the right ventricle, than over the left ventricle. This shows that the muscular power of the latter is diminished more than that of the former, a fact which is highly significant of softening, because the causes inducing merely functional weakness are alike operative on the two ventricles.[1] Finally, the reduction of the pulse in frequency below the normal average, which is observed at the time of convalescence, in a certain proportion of cases of typhus and typhoid fever, is supposed by Dr. Stokes to denote that softening has taken place.

Treatment of Softening of the Heart.

The occurrence of softening of the heart in the course of typhus and typhoid fever furnishes an additional indication for sustaining measures, viz: diffusible or alcoholic stimulants, and concentrated nutriment. The tendency to death by asthenia is enhanced by the complication of this lesion, and, hence, the means of obviating this tendency are to be pushed with more vigor whenever there are reasonable grounds for supposing that the lesion has taken place. These remarks are equally applicable to the treatment of softening when it occurs in other pathological connections, as in the eruptive fevers, pyemia, &c. Dr. Stokes has called attention to the importance of the physical signs of cardiac weakness in determining the extent to which sustaining measures, and especially alcoholic stimulants, are indicated in the treatment of fevers. The diminished intensity or suppression of the first sound of the heart, together with its alterations in duration and quality, constitute a better criterion of the loss of muscular power which the organ has sustained, than the pulse or other symptoms. In fact, the pulse, as stated already, does not always represent fairly the force with which the left ventricle contracts. The evidence obtained by auscultation and palpation is more reliable. With reference to this end, physical exploration practised from day to day during the

[1] It may be conjectured that when softening is limited to or especially marked in the left ventricle, the pulmonary second sound will be more intense than the aortic. This is a point to be settled by clinical observation.

febrile career, is of great practical value, and is too much neglected by medical practitioners. In endeavoring to decide between softening and functional debility of the heart, there may be often room for doubt, but, happily, so far as relates to treatment, the indications are the same in both cases, and, consequently, no harm results from error in this differential diagnosis. Practically, the important end is to estimate correctly, by means of the physical signs, the degree of cardiac weakness.

INDURATION OF THE HEART.

Induration of the muscular walls of the heart, sufficient to constitute in itself a lesion or an important element of organic disease, is extremely rare. Pathologically it involves different conditions. The muscular tissue appears to be capable of becoming condensed in a remarkable degree without obvious disorganization or morbid deposit. An instance was described by Corvisart in which the heart, when struck, sounded like a dice-box or hollow horn vessel, and yet the natural appearance of the muscular substance was preserved. The microscope had not then been brought to bear on the study of minute anatomy. Portions of the walls present sometimes the firmness and appearance of cartilage. This must proceed either from exudation due to an inflammatory process, or from hypertrophy of the cardiac fascia, described by Dr. Robert Lee,[1] which forms a sheath around the arteries, veins, and nerves contained within the substance of the heart. Calcareous deposit sometimes exists between the muscular fibres, here as elsewhere due to the metamorphosis of exudation matter. In a case described by Burns, the ventricles were said to be so completely ossified as to resemble the bones of the cranium. Some allowance is doubtless to be made for exaggeration in this comparison. These conditions probably belong among the results of inflammation. In a practical point of view, induration of the cardiac walls claims only a passing notice. Not only is it extremely rare, but it is wanting in distinctive signs and symptoms. The diagnosis is impossible. It may be stated as

[1] Philosophical Transactions, 1848. *Vide* Bellingham on Diseases of the Heart, part i., Dublin, 1853, p. 24.

a rule applicable, at least, to diseases of the heart, that the difficulty of diagnosis is inversely in proportion to its practical importance. This lesion affords an illustration of the rule. Induration proceeding from either of the conditions mentioned, is irremediable. It was conjectured by Laennec that the heart-sounds would be intensified by an indurated state of the walls, so as to be heard at a distance from the chest in some cases. Clinical observation, however, has shown, on the contrary, that the sounds are enfeebled. This would be expected in view of our present knowledge of the mechanism of the sounds.

CARDIAC ANEURISM.

The term aneurism was formerly applied to enlargement of one or more of the compartments of the heart, due either to hypertrophy or dilatation. This application of the term is manifestly inappropriate, and is now discontinued by most writers. Cardiac aneurism is properly a circumscribed or pouch-like dilatation occurring in one or more of the anatomical divisions of the organ. In the great majority of instances it is seated in the left ventricle. It does not occur in the right ventricle, but, in a very small proportion of cases, it has been observed in the left auricle. It is an extremely rare lesion, yet Mr. Thurman was able to collect for analysis, from various sources, accounts of seventy-four cases.[1] The aneurismal dilatation forms a tumor varying in size in different cases from that of a small nut to a sac as large as the heart itself. It contains layers of condensed fibrin and various forms of coagula, like arterial aneurisms. It is sometimes lined or studded with calcareous matter. It occurs in the great majority of cases at the apex, but it may be situated at any point on the anterior or posterior surfaces of the ventricle, and on the inter-ventricular septum. The cavity of the sac communicates with the ventricular cavity by an aperture varying in different cases as respects form and size, being sometimes

[1] Mr. Thurman's paper was published in the Medico-Chirurg. Transactions, London, vol. xxi., 1838. The reader will find an abstract of it in Hope's Treatise on Diseases of the Heart, etc., Am. ed., 1842, p. 313. Also in Bellingham on Diseases of the Heart, part ii., 1857. In the latter, the reader will find copious references to the literature of the subject.

direct and in some cases sinuous. The walls of the sac, in some cases, include the endocardial and pericardial membranes unbroken, the muscular substance having mostly or quite disappeared; or there has occurred solution of continuity of the endocardial membrane. In the latter case, according to Rokitansky, the aneurism may be considered as an *acute*, and in the former as a *chronic* affection. When the endocardial membrane is perforated, the lesion probably commenced by disease of this membrane, the other tissues undergoing dilatation from the pressure of the blood. This is analogous to the false aneurism of surgical writers. The tumor, under these circumstances, does not attain to a great size. In chronic cases, without perforation of the endocardium, the walls of the heart yield to the pressure of blood and become dilated in consequence of a morbid condition at the part affected. This condition generally results from circumscribed inflammation, and consists of softening, or the substitution of new structure for the muscular tissue. The affection, when thus induced, is analogous to the true aneurism of surgical writers. It is supposed that a circumscribed abscess of the walls of the heart, opening into the cavity of the ventricle, may lead to aneurism. This is a very brief synopsis of the views held by pathologists concerning the formation of cardiac aneurism. For a fuller consideration of the subject, the reader is referred to works on morbid anatomy.[1] Two or more aneurismal tumors are sometimes present in the same case. The affection occurs much oftener in the male than in the female.

Cardiac aneurism may be associated with enlargement of the heart by hypertrophy or dilatation, and with valvular lesions, but these affections do not uniformly exist, although present in a large proportion of cases. In the cases analyzed by Mr. Thurman, valvular lesions coexisted in ten, and were stated not to exist in eight, the whole number of cases of aneurism of the left ventricle being fifty-eight. In ten cases only of the whole number, *i. e.*, of fifty-eight, was the absence of hypertrophy or dilatation stated. Adhesion of the pericardial surfaces over the tumor takes place in some instances, and is wanting in others. Aneurismal dilatation of the mitral valve will be more properly noticed under the head of valvular lesions.

Aneurism of the heart may end in rupture and sudden death,

[1] Rokitansky treats of this subject at considerable length. An abridgment of Rokitansky's views is contained in the work by Jones and Sieveking.

the blood being poured into the pericardial sac, provided adhesion of the free surfaces of the pericardium have not taken place, and, if so, the opening may take place into the left pleural cavity. But, prior to the occurrence of this event, a fatal result may take place in consequence of the embarrassment of the circulation occasioned by the tumor, and by the concomitant lesions with which it is generally associated.

The existence of this affection is not determinable during life. In no case as yet observed, has the diagnosis been made, nor is it probable that any diagnostic criteria will be ascertained by farther clinical observation. The symptoms in the cases which have been reported, are those denoting some grave cardiac affection, but they are generally due, in a great measure, to coexisting valvular lesions or enlargement, or to both. Nor are the physical signs more distinctive. The passage of blood currents into and from the sac, is likely to give rise to a murmur, which may accompany either sound of the heart, or both sounds. A friction murmur may also be produced. But there are no circumstances which can possibly lead the diagnostician to pronounce that these signs are due to an aneurismal tumor. He may be led to suspect this affection, but he is never justified in deciding with any positiveness that it exists. The circumstances favoring such a suspicion, are those which show that some anomalous form of disease is present. For example, as remarked by Hope, an endocardial murmur may be found which is not referable to the arterial or auricular orifices by the rules of localization to be hereafter considered. Valvular lesions, as the source of the murmur, being thus excluded, and the murmur being evidently due to some organic affection, the hypothesis of cardiac aneurism is admissible; but intra-ventricular murmur is sometimes produced, not referable to the orifices, and, on the other hand, aneurismal dilatations do not always give rise to murmur. So a pericardial or friction murmur may proceed from various conditions, irrespective of present pericarditis. In short, the diagnosis is necessarily unattainable. The affection is one of the rare forms of disease which give rise to more doubt and difficulty, the better acquainted the practitioner is with the diagnostic signs and symptoms of cardiac lesions. He may be aware that he has to deal with some anomalous affection, but he is unable to determine its character. Here, as in other similar instances, the inability to arrive at the diagnosis is not, in a practical point of view, to be deplored; for, were the existence of cardiac aneurism determinable, the treatment would

be that which is indicated by the symptoms without this knowledge. The lesion is irremediable, and the measures best suited to retard the dilatation and prolong life, are those which are applicable to cases of valvular lesions and enlargement, with which the affection is often associated.

RUPTURE OF THE HEART.

Spontaneous rupture of the heart is a lesion of very rare occurrence. It may fairly be doubted if it has ever occurred as a result purely of the violent muscular activity of the organ. In a physical point of view, a broken heart is a poetical license, exclusive of the cases in which the event is dependent on some prior morbid condition of the cardiac parietes. It is an accident incidental to different local affections. In the great majority of cases, it takes place in consequence of softening from fatty degeneration. It may follow extravasation into the muscular substance, constituting the condition called by the French writers apoplexy of the heart, which has been investigated fully by Cruveilhier; great attenuation of the walls in some cases of dilatation; circumscribed abscess; ulcerative perforation of the endocardium; and softening from inflammation. The seat of rupture, in the vast majority of instances, is the left ventricle, either on the anterior or posterior surface. Statistics differ as to the relative liability of the two surfaces. It has been observed in the auricles as well as in either ventricle. Usually a single opening takes place, varying in size from a very minute aperture to a rent of considerable size; but instances have been reported of rupture simultaneously at several different points. It occurs oftener in the male than in the female, and almost always at an advanced period of life. The coexistence of hypertrophy or of aortic obstruction favors its occurrence. It may be attributable to some unusual muscular exertion acting as an exciting cause, but in a large proportion of the reported cases, the patients were in a state of repose when it took place.[1] The only instance of rupture which has fallen under my observation occurred in a patient admitted into

[1] *Vide* paper by Dr. Hallowell, of Philadelphia, giving an analysis of thirty-four cases, in the *American Journal of Medical Sciences*, 1835. For copious references to the literature of this subject see Bellingham, *op. cit.*, part II., 1857.

the Charity Hospital at New Orleans with delirium tremens. I did not see the patient during life. He died suddenly and unexpectedly, no affection of the heart having been suspected. On examination after death, a rent was found at the upper and anterior part of the right ventricle near the pulmonary artery. The inner layer of muscular fibres was torn over a space wider than the external opening, showing the gradual progress of the disruption from within. The heart was enlarged, weighing a fraction over fourteen ounces. The ventricular walls were not increased in thickness. The right ventricle was covered with fat, and the walls presented both the gross and microscopical characters of advanced fatty degeneration. At certain points, fatty matter appeared to have replaced the greater part of the ventricular walls, the muscular tissue being reduced to a thin layer, not more than a line in thickness. The patient was about sixty-five years of age. The previous history of the case was not ascertained.

Rupture of the heart is almost inevitably fatal, and death generally follows at once. In some instances, however, life has continued for several hours. The aperture in these instances was quite small, or the escape of blood was retarded by the formation of a coagulum at the point of rupture. A repair of the solution of continuity is perhaps not impossible, although infinitely improbable. Dr. Walshe states that one case has been recorded of death from rupture in which a former rupture was discovered, firmly filled by a fibrinous coagulum adherent to the wall of the heart. The mechanism by which the fatal result is produced has given rise to considerable discussion. Blood is poured into the pericardial sac with more or less rapidity according to the extent of the rupture. But this sac will not contain sufficient liquid for death to be referred to the hemorrhage alone. Paralysis of the heart from the mechanical compression of the accumulation of blood within the pericardial sac is doubtless an important agency.

Time and opportunity are seldom offered for an investigation with reference to diagnosis. If life be prolonged for some hours, the symptoms are those which denote syncope with præcordial distress, and coma may ensue before dissolution. Happily here, as in other instances in which a positive diagnosis is unattainable, it would not, if attainable, affect the treatment. The indications derived from the symptoms alone are those which would be furnished by the knowledge of the accident which has occurred. Death occurring suddenly, or a few hours after the sudden develop-

ment of alarming syncope, in a person advanced in years, who had previously presented evidence of cardiac disease, and especially of fatty degeneration, warrants a strong suspicion of rupture.

Rupture of the valves of the heart, or of the tendinous cords and papillary muscles, falls more appropriately under the head of valvular lesions than in the present connection.

In addition to the lesions affecting the walls of the heart, which have been considered in this chapter, there are some others, extremely rare and unattended by any distinctive symptoms or signs, and, therefore, of little interest or importance in a practical point of view. Carcinomatous and tuberculous deposits have been known to extend from beneath the endocardial and pericardial membranes more or less into the muscular substance of the organ. Few organs of the body, however, are more exempt from these heteromorphous formations than the heart. So slight is the probability of their existence in a given individual case, that they are scarcely to be taken into account in the investigation of cardiac affections which are evidently anomalous. The presence of these deposits in other parts of the body may constitute a slight ground for suspicion that they have invaded the heart, if the signs and symptoms show that the organ is affected with some indeterminable form of disease. In this category are to be included extravasation of blood, or cardiac apoplexy, to which allusion has been already made, and cysts containing entozoa.

CHAPTER III.

LESIONS AFFECTING THE VALVES AND ORIFICES OF THE HEART.

Aortic lesions—Mitral lesions—Primary effects of valvular lesions on the circulation—Points to be observed in post-mortem examinations—Pathological processes involved in the production of valvular lesions—Symptoms and secondary pathological effects of lesions affecting the valves and orifices of the heart—Symptoms and pathological effects referable to the heart—Enlargement of the several portions of the heart in relation to mitral, aortic, tricuspid, and pulmonic lesions, respectively—Pain, palpitation, the pulse, venous turgescence, and pulsation—Symptoms and pathological effects referable to the circulation—Cardiac dropsy—Arterial obstruction by fibrinous deposits detached from the valves or orifices of the heart (embolia)—Symptoms and pathological effects referable to the respiratory system: Dyspnœa, cardiac asthma, cough, muco-serous expectoration and hæmoptysis, pulmonary apoplexy and œdema, bronchitis, pneumonitis, pleurisy, and emphysema—Symptoms and pathological effects referable to the nervous system: Apoplexy, paralysis, arterial obstruction, defective supply of blood to brain, pseudo-apoplexy, cephalalgia, vertigo, tinnitus aurium, etc., sleep, mental condition—Symptoms and pathological effects referable to the digestive system and nutrition: Hepatic congestion, nutmeg liver, portal congestion, enlargement of liver, cirrhosis, indigestion, hæmatemesis, enterorrhœa, melæna, hæmorrhoids, enlargement of spleen, nutrition—Symptoms and pathological effects referable to the genito-urinary system: Congestion of kidneys, diminished secretion of urine, albuminuria, structural degenerations of kidney, or Bright's disease—Generative functions—Symptoms and pathological effects referable to the countenance and external appearance of the body: Lividity, expression, anæmia, capillary congestion, erythema, bloodless fingers.

LESIONS of the valves or orifices of the heart, or *valvular lesions*, as they are concisely called, are present in a very large proportion of the cases of organic disease of this organ which come under the cognizance of the physician. In addition to the intrinsic interest which they possess as subjects for clinical study, they are important as standing in a causative relation to other cardiac lesions, more especially enlargement of the heart, and also as giving rise to pathological effects manifested in other parts of the body. They are important as sustaining a relation of dependence to other diseases, particularly acute rheumatism, a relation which has been established by modern researches. In connection with physical signs, and as exemplifying the wonderful precision of diagnosis which has resulted from the application of auscultation within the

past few years, the clinical study of these lesions is highly interesting. Inquiries with respect to their origin and mode of production involve pathological points of much interest and importance. To the latter, brief reference will alone be made, a full discussion of them being inconsistent with the practical objects of this work. The various morbid appearances incidental to the lesions will be summarily considered, a lengthened description belonging more appropriately to works on pathological anatomy. In treating of valvular lesions, the main objects will be to show their immediate and remote effects, the symptomatic phenomena to which they give rise, their physical signs and diagnosis, and, finally, the indications for treatment. The physical signs of these lesions consist of abnormal modifications of the natural heart-sounds, and also of superadded adventitious sounds distinguished as *murmurs*. The importance of the latter, and the various considerations connected with their diagnostic application, require that they should be treated of at some length.

Lesions of the valves and orifices of the heart, exclusive of congenital malformations, are seated as a rule in the left half of the organ; that is to say, in the great majority of cases they are either mitral or aortic. The tricuspid and pulmonic valves and orifices rarely become affected after birth. Still more unfrequently do the latter present extensive structural alterations such as are often found in the corresponding situations in the left half. When they occur, they are generally, but not invariably, associated with mitral and aortic lesions. It is a curious fact that the lesions of fœtal life, giving rise to the congenital malformations which will be noticed in a subsequent chapter, affect by preference the right half of the heart, reversing the rule which obtains after birth. The changes which the valves and orifices present in different cases, vary greatly in degree and kind. The morbid appearances are exceedingly diversified. As before remarked, a full description of these belongs more appropriately to the works on pathological anatomy, and to these the reader is referred.[1] I shall content myself with a simple enumeration of the more prominent forms or varieties, considering the aortic and the mitral lesions under distinct heads. Pulmonic and tricuspid lesions will be noticed in connection with the pathological effects of valvular lesions, referable to the heart, and also in treating of congenital malformations.

[1] Rokitansky's great work, or Jones and Sieveking, may be consulted for this purpose.

Aortic Lesions.—Lesions may be confined to one or two of the semilunar segments; but in general all are more or less affected, although rarely in an equal degree. The segments may be simply thickened and somewhat contracted. If the contraction be not enough to render them insufficient, that is, permitting regurgitation, the thickening only renders their action less free than in health. One or both the surfaces may present vegetations or excrescences, varying in size from a pin's head to a pea or bean. These are frequently situated on or near the free extremity of the segments. I have seen in one case masses resembling fibrin attached to the lower surface, as large collectively as a walnut, hanging downward an inch within the ventricle. These vegetations are sometimes easily detached, so easily as to render it altogether probable that they are sometimes washed away by the current of blood during life. In other instances they are firmly attached. They must, in proportion to their number and size, embarrass the movements of the valve. Morbid growths of cartilaginous hardness, and calcareous deposits are often found situated at the attached margins of the semilunar segments, extending partially or entirely over them. These render the segments more or less rigid and permanently expanded. One or more of them may be thus affected. In proportion to the amount of morbid material, and the space which the expanded segment or segments occupy, will the size of the arterial orifice be diminished, and the current of blood broken and interrupted. Occasionally the segments become united at their sides, and, remaining expanded, the orifice is diminished to a small aperture. I have seen it as small as a crow's quill; it has been observed even considerably smaller than this, so as hardly to admit the passage of a fine probe. One or more of the segments may be expanded and crumpled, being bent either upward or downward. They are sometimes greatly shrivelled or corrugated, leaving a permanently open aperture of greater or less size. The partition between two of the segments is occasionally wanting, fusion into a single segment having taken place. It is sometimes difficult to say whether this is due to disease after birth, or a congenital malformation. Attenuation of the segments is another variety of lesion, a species of atrophy, and in this condition they are liable to become perforated or cribriform.

Finally, rupture may take place in different directions. A segment may be torn vertically from the free margin toward the base; it may be partially torn away from its attachment, or there may be

one or more fissures at the base, the lateral ends remaining attached. These different varieties of lesion are by no means observed separately in different cases, but they are usually to a greater or less extent combined in the same case.

Mitral Lesions.—These are essentially the same as the aortic lesions, the points of difference relating chiefly to the different form and arrangement of the mitral valve. They consist of thickening and contraction of the valvular curtains; rigidity from calcareous deposit; attenuation and perforation; adhesion of the sides of the two curtains, giving rise to a funnel-shaped canal from the auricle to the ventricle, opening into the latter by a slit or small aperture; adhesion of the curtains to the walls of the heart; shortening and, in some instances, cretaceous hardness and brittleness of the tendinous cords; accumulation of masses of calcareous matter at the base of the curtains, diminishing the size of the auriculo-ventricular orifice, and presenting an irregular surface to the current of blood; rupture of the curtains in various directions and of the tendinous cords; warty vegetations or excrescences and deposit of fibrin in masses of variable form and size, adhering loosely or firmly, etc. This valve is subject to circumscribed dilatations called aneurisms, which form pouches varying from the size of a pea to that of a walnut, protruding into the cavity of the auricle, and containing coagula or laminated fibrin. These aneurismal dilatations present, in some instances, all the membranous structures of the valve unbroken, and, in other instances, perforation of one of the endocardial laminæ, the distinction between false and true aneurisms being thus maintained here as in aneurismal dilatations of the cardiac walls. This variety of lesion is of rare occurrence.

The foregoing summary of the various lesions affecting the valves and orifices of the heart is intended merely to refresh the memory of the reader. In order to form a proper idea of the great diversity of morbid appearances, it is important to consult works on morbid anatomy in which they are fully described and illustrated, and, as far as practicable, also to examine morbid specimens. The immediate pathological importance of the lesions depends on the primary effects which they produce on the blood-currents. Arranged with reference to these effects, they may be distributed into three classes, to wit: *First*, as involving obstruction to the

onward or direct blood-currents; *second*, as inducing insufficiency of the valves and allowing regurgitation or retrograde currents; and, *third*, as interfering more or less with the freedom of the action of the valves and roughening surfaces which are normally smooth and polished, but without giving rise to either obstruction or insufficiency. Of these three divisions, according to the primary effects of the lesions, the two first alone possess much *immediate* pathological importance. The last class of lesions affect the blood-currents but little or not at all. Their pathological importance is remote, that is, it relates to a prospective period when, by ending in more serious changes, they may induce either obstruction or insufficiency. In the clinical study of valvular lesions, however, it is highly important, as will hereafter appear, to bear in mind the fact that abnormal conditions may exist which are not of immediate pathological importance, inasmuch as they do not involve either contraction of the orifices or regurgitation, but which give rise to physical signs. To this important fact reference will again be made.

The primary effects of valvular lesions on the circulation, which are of a nature to possess immediate pathological importance, are, then, produced by means of obstruction and insufficiency. Other things being equal, the degree of importance belonging to these effects is proportionate to the amount of obstruction and insufficiency. Obstruction may exist without insufficiency, and *vice versâ*, but it often happens that the lesions are such as to occasion both at the same time. These remarks apply indifferently to aortic and to mitral lesions. Now, aortic lesions may exist without mitral lesions, and *vice versâ*, but frequently lesions are present, in the same case, in both situations. Cases are greatly diversified by the different combinations of aortic and mitral lesions and their primary effects. Thus, there may be lesions of either the aortic or mitral orifice separately, which involve insufficiency without obstruction; the lesions in either situation may occasion obstruction without insufficiency; obstructive aortic lesions may be associated with regurgitant mitral lesions, or *vice versâ;* there may be obstruction, or, on the other hand, regurgitation, both at the aortic and mitral orifice, and, finally, aortic and mitral lesions may coexist, each involving both obstruction and insufficiency. These various combinations would seem to render the clinical study of the valvular lesions extremely complicated, but the application to this study of physical exploration has rendered it practicable, in most cases, to determine whether

aortic or mitral lesions exist separately or combined, whether obstruction or regurgitation, or both, are produced by existing lesions, and to estimate the amount of damage which the heart has sustained. The reader will be better able to judge of the correctness of this statement after the physical signs and diagnosis have been considered.

As regards relative frequency in the occurrence of mitral and aortic lesions, in my own experience, the former slightly preponderate. Of 104 recorded cases, in 40 the lesions were mitral, and in 37 aortic. In 14 of these cases mitral and aortic lesions coexisted, and in 4 cases only were the tricuspid or pulmonic valves the seat of lesions. Of 367 cases analyzed by Dr. T. K. Chambers, the mitral and aortic valves were affected with thickening, contraction, or morbid deposit in 121; the aortic valves were affected alone in 107; the mitral in 96; the mitral and tricuspid valves in 10; the mitral, aortic, and tricuspid in 10; the four sets of valves in 9; the tricuspid alone in 1; the tricuspid and aortic in 2; the aortic, mitral, and pulmonary in 2; the tricuspid and aortic in 2; and the aortic and pulmonary valves in 4.[1]

In determining the pathological importance of lesions at post-mortem examinations, the points for observation are embraced in the following questions: Is there contraction of one or more of the orifices, and, if so, to what extent? Are the arterial or auricular valves sufficient to protect the orifices, and, if not, how great is the insufficiency? These points are to be settled by laying open the cavities and carefully examining the orifices and valves. With a proper knowledge of the normal appearances, the existence or otherwise of contraction or insufficiency may be readily ascertained by the eye, and the amount of obstruction or regurgitation estimated accurately enough for practical purposes. The sufficiency or insufficiency of the aortic valve may be ascertained, before laying open the cavities and vessels, by resorting to the water test. This test consists in suspending the heart by hooks introduced into the aorta above the valves, having first tied the coronary arteries and opened the left ventricle by slicing off the apex of the organ, and then pouring a stream of water into the aorta. If the valve be sufficient, little or no water passes into the ventricle; but if there

[1] *Decennium Pathologicum.* Brit. and For. Med.-Chir. Rev., vol. xii., 1853. In these cases were probably included cases of congenital malformation. The valves were affected in 367 of 2161 bodies examined.

be insufficiency, the water escapes more or less freely from the opening at the apex. This test is far less reliable in its application to the mitral valve. It is applied to this valve by suspending the heart with the apex upwards, the left ventricle having been opened, and the aorta and coronary arteries tied. If water poured into the opening at the apex do not pass into the left auricle, the mitral valve is sufficient. This test demonstrates sufficiency in a certain number of cases. But if the water pass into the auricle, it does not follow that the mitral valve was insufficient during life, the conditions being so widely different in this experiment. If the orifices are contracted so as to oppose an obstacle to the blood-current, it is obvious to the eye, provided the observer have been accustomed to examine hearts in which the size of the orifices is normal. In recording post-mortem observations, it has been customary to note how many fingers may be passed readily through the orifice. This is a rough method of measurement, but in most instances it is sufficiently precise. Greater accuracy, of course, is obtained by actual measurement either of the diameter or circumference. As standards of comparison, the average size in healthy hearts is to be determined. The numerous measurements by Bizot give the following mean results: The average circumference of the mitral orifice in the adult male is about four inches. The long diameter, according to Dr. Bellingham, is about one inch. In the female, the size is somewhat less. The average circumference of the aortic orifice in the adult male is three inches; the diameter, according to Dr. Bellingham, is about an inch. In the female, the size is somewhat less. The tricuspid orifice is somewhat larger than the mitral, in health; and this is true of the pulmonic orifice prior to the age of fifty. The observations of Bizot show that the orifices, as well as the heart itself, increase in size gradually from birth to old age.

What pathological processes are involved in the production of valvular lesions? This question claims a few words before passing to consider the symptoms and pathological effects of these lesions. It opens up a broad field, embracing mooted topics, the discussion of which does not fall within the scope of this work. I shall present very briefly the general views which appear to be most consistent with our present knowledge, and which are of importance in their practical bearings. Thickening of the valves, the accumulation of fibrinous matter in the form of vegetations or excrescences, and calcareous concretions, are due to abnormal deposit, which may either take place as an exudation from the

bloodvessels or be derived from the blood contained within the heart-cavities. It is certain that both of these sources of deposit are concerned in the changes referred to. When fibrinous, calcareous, or other matter is situated beneath the endocardial membrane, it must be an exudative deposit. An organized growth is to be considered as exudative. On the other hand, it is sufficiently well ascertained that more or less of the deposit, in certain instances, proceeds from fibrin attracted from the blood exterior to the vessels, *i. e.*, within the chambers of the heart. Roughness of the endocardial membrane conduces to deposit from this source. This is illustrated by Mr. Simon's experiment of passing a thread through an artery;[1] fibrin coagulates and adheres to the thread, presenting an appearance not unlike the so-called vegetations sometimes observed on the valves. It is by no means probable that these fibrinous concretions are organizable.[2] There is reason to suppose that their production is favored by certain conditions of the blood, such as a superabundance of the fibrinous element, which occurs in various affections, and especially in acute rheumatism. The deposit from either source undergoes metamorphoses, produced by the elimination of certain constituents and the addition of others. Thus, a deposit primarily fibrinous, may become, in the course of time, calcareous. The contraction or shortening of valves, their rigidity and permanent expansion, crumpling, rupture, etc., are ulterior results of deposit and the metamorphoses which it undergoes. Attenuation and cribriform perforations are due to defective nutrition or atrophy, which may render the valves unable to sustain the pressure of the blood, and give rise to laceration. By a perversion of nutrition, also, according to Rokitansky, a gelatinous substance is sometimes substituted for the fibrous tissue of the valves, rendering them weak and liable to rupture. To what extent is inflammation involved in these processes? Post-mortem examinations, when death has occurred during or shortly after an attack of acute endocarditis, have shown that the inflammation affects more especially the valves, and leads to deposit both by exudation and coagulation. Moreover, clinical observation shows that, in the great majority of the cases of valvular lesions, the persons affected have, at some former period of their lives, expe-

[1] Lectures on General Pathology.

[2] *Vide* Ch. Robin in Dictionnaire de Médecine, par Nysten, 1854; also Chimie Anatomique, par Robin et Verdeil.

rienced one or more attacks of acute rheumatism, a disease which clinical observation, within late years, also has shown to be complicated with endocarditis in a large proportion of instances.[1] From these facts it may be logically inferred that valvular lesions originate most frequently in endocardial inflammation. The immediate local effects of endocarditis, as will be seen when we come to treat of that affection, are generally unimportant. During the progress of the endocarditis, and, perhaps, for many years afterwards, there may be no obvious symptoms denoting cardiac lesions. The structural changes which these effects gradually induce, at length give rise to obstruction or regurgitation, or both, and, finally, symptoms are developed which point to the heart as the seat of disease. When, thus, inflammation constitutes the first step in the production of lesions of structure, it is a by-gone and remote event at the time these lesions have become of immediate pathological importance; it has long before ceased to be an active element of the cardiac affection, its products, with their metamorphoses, and the changes induced by them, having, by degrees, led to the existing morbid condition of the organ. But it is not probable that the lesions originate always in inflammation. An exudation similar to that which constitutes the atheroma of arteries is apparently the first event in some cases. This cannot be considered as involving inflammation, unless the term be so defined as to embrace every process attended by a solidifying deposit. There is no ground to suppose that attenuation or atrophy, and the gelatinous degeneration described by Rokitansky, are dependent on an inflammatory action.

[1] Of *sixty-one* cases of valvular lesions, in the histories of which the existence or non-existence of rheumatism at a former period of life is noted, this affection had occurred in *forty-three*. Rheumatism had existed in a larger proportion of the cases of mitral than of aortic lesions. Of *twenty-nine* cases of mitral lesions, rheumatism had occurred in *twenty*. Of *fourteen* cases of aortic lesions, rheumatism had occurred in *seven*. The cases of aortic and mitral lesions, however, presented the largest proportion in which rheumatism had existed. Of *eighteen* cases, this affection occurred in *sixteen*.

SYMPTOMS AND SECONDARY PATHOLOGICAL EFFECTS OF LESIONS AFFECTING THE VALVES AND ORIFICES OF THE HEART.

The primary effects of valvular lesions, which are of immediate pathological importance, have been already considered. They are, obstruction to the passage of blood by contraction of the orifices, regurgitation or the flow of blood in a retrograde direction owing to insufficiency of the valves, these effects being produced either separately or conjointly. Hence, the lesions affecting the valves or orifices may be distinguished as *obstructive* or *regurgitant* lesions; and as all the valves or orifices of the heart may be affected either separately or in various combinations, valvular lesions may be divided after their seat and primary effects into obstructive and regurgitant lesions, situated respectively, at the mitral and aortic orifices, and, much more rarely, at the pulmonic and tricuspid orifices. The secondary or remote pathological effects of these lesions, for the most part, are traceable to the primary effects. The disturbance of the circulation, due to cardiac obstruction and regurgitation, singly or combined, gives rise to a great number and variety of morbid conditions and manifestations intrinsically more or less serious, and important, also, as symptoms of the heart affection. It will be most convenient to arrange these ulterior effects according to the different anatomical systems in which they occur. Pathological effects of much importance are produced in the heart itself; other effects are appropriately considered as pertaining to the vascular system, not being limited in their consequences to any particular situation; others relate respectively to the respiratory, nervous, digestive, genito-urinary systems, etc. In considering the effects after this arrangement, their relations to obstructive and regurgitant lesions seated at the different orifices will be incidentally considered.

Symptoms and Pathological Effects referable to the Heart.

Valvular lesions involving obstruction or regurgitation, sooner or later, in the great majority of cases, lead to enlargement of the heart. They lead to this result by inducing over-distension of the cavities and over-excitement of the organ, as has been con-

sidered in the chapter devoted to the subject of enlargement. The enlargement may be due either to predominant hypertrophy or dilatation. The latter predominates in most instances in which the cardiac disease has existed for a long period, and proved directly fatal, *i. e.* when death is not attributable to an intercurrent affection. The hypertrophy or dilatation is generally marked in, and may be limited to certain portions of the heart. The enlargement commences at one of the ventricles or auricles, according to the situations of the valvular lesions, and thence extends successively over the other compartments, observing a general rule of extension, exceptions to the rule, however, occurring not unfrequently.

Obstructive or regurgitant lesions at the mitral orifice induce, as a rule, first, dilatation of the left auricle; next, dilatation or hypertrophy of the right ventricle; next, dilatation of the right auricle, and finally, in most cases, more or less enlargement either by hypertrophy or dilatation of the left ventricle. This is the regular order of effects upon the heart, the mechanism of which has been already described. Variations from this rule are frequently observed. Thus the right auricle is sometimes much more dilated than the left, when the valvular lesions are exclusively mitral; and occasionally under these circumstances, the left ventricle is found to be more enlarged than the right. In these exceptional instances, either the walls of the portions which are enlarged out of the natural order, are particularly prone to enlargement, or there exists causes superadded to the valvular lesions. Thus, emphysema, coexisting with mitral lesions, will cause the enlargement of the right ventricle and auricle to preponderate much more than if the mitral lesion existed alone. In some cases, superadded causes may exist remote from the heart, which are not readily ascertained, causing enlargement of the left ventricle to preponderate, when, as a result of mitral lesion alone, this should be the cavity last and least affected. As an exceptional occurrence the left ventricle is sometimes diminished in size when, in consequence of mitral lesions, the other portions are enlarged. This fact, first pointed out by Dr. Law, of Dublin, is explained by the diminished supply of blood received by that ventricle when there exists much obstruction of the mitral orifice. The enlargement of one ventricle may be by hypertrophy, and that of the other by dilatation. Thus the right ventricle may be hypertrophied and the left dilated, or *vice versâ*. The amount of enlargement of the heart, as a whole, varies greatly in different cases, and what is remarkable, is not proportionate to the amount

of obstruction or regurgitation, a fact which shows the influence of causes subsidiary to the valvular lesions. Very great enlargement is found associated with lesions involving only moderate obstruction or regurgitation, and, on the other hand, the heart is sometimes found to be but little, or not at all enlarged, when there exists a marked degree of contraction or insufficiency. The mitral orifice has been reduced to the size of a crow's quill, without notable enlargement of any of the cavities.[1] This fact also shows the importance of causes superadded to valvular lesions. As a rule, contraction of the mitral orifice, in other words, obstruction, tends to give rise to enlargement, more than insufficiency or regurgitation; but the tendency is of course greater when, as is frequently the case, contraction and insufficiency are conjoined. The latter occurs in the instances in which the curtains of the mitral valve become adherent at their sides, leaving a funnel-shaped canal opening into the ventricle by a narrow fissure resembling a button-hole or the chink of the glottis.

Enlargement proceeding from aortic lesions invariably commences at the left ventricle. If the valvular lesions are exclusively aortic, this ventricle is always enlarged disproportionately to the other portions of the heart, and the enlargement may be limited to the left ventricle. An examination of the heart before the cavities are opened often suffices to show that the valvular lesions are probably aortic. Either hypertrophy or dilatation may predominate in the enlargement proceeding from these lesions. As a rule, if the lesions are of a nature to allow of regurgitation without producing obstruction, dilatation predominates; but if the lesions produce obstruction without regurgitation, hypertrophy is marked. This rule is not without exceptions, but it holds good in the great majority of cases. Thus, of 21 cases of either regurgitation or obstruction, the notes of which are before me, 3 only were exceptional. Of these 21 cases, in 13 there existed regurgitation without contraction, and in 2 cases hypertrophy was predominant, dilatation predominating in the others; in 8 cases there was obstruction without regurgitation, and in all save one hypertrophy was predominant. Aortic lesions, however, frequently give rise both to obstruction and regurgitation, and in proportion as the one or the other preponderates, dilatation or hypertrophy will be likely to be marked.

[1] The cabinet of the Boston Society for Medical Improvement contains two specimens, illustrative of this statement, *vide* printed catalogue, pages 73 and 86.

Usually the enlargement extends to the other portions of the heart. The right ventricle is not proportionately enlarged, unless there are concurrent causes which exert their effect especially on this ventricle. Pulmonary emphysema, coexisting with aortic lesions, may render the enlargement of the right ventricle as great, or even greater, than that of the left. Of the two auricles, the tendency of aortic lesions is to dilate, first and especially, the left, but in some instances dilatation of the right is more marked. Enlargement associated with aortic, as well as with mitral lesions, is by no means in all cases proportionate, as regards amount, to the degree of obstruction or regurgitation. Enormous enlargement is observed in cases in which the contraction or insufficiency is small, and, on the other hand, in some instances in which the obstruction must have been extremely great, the size of the heart has been found slightly or not at all increased. This fact is illustrated by a specimen contained in the cabinet of the Boston Society for Medical Improvement, the aortic orifice being so much contracted as hardly to admit of the passage of a small probe.[1] These facts here, as in the case of mitral lesions, show the importance of concurrent causes or morbid conditions in determining the amount of enlargement of the heart.

When, as is frequently the case, mitral and aortic lesions are associated, involving, in each situation, either obstruction or regurgitation, or both, the effects of the two classes of lesions are conjoined. Other things being equal, the enlargement of the heart, as a whole, is proportionately greater under these circumstances. The aortic lesions give rise to enlargement of the left ventricle, and combine with the mitral lesions in leading to enlargement of the other portions of the heart. Among cases of this description we are likely to find examples of excessive augmentation of bulk, constituting the *cor bovinum* of the old writers.

The pulmonic and tricuspid valves, as already stated, are rarely the seat of those structural changes which so often affect the valves of the left side of the heart. Valvular lesions, seated in the right side when they occur, are usually, but not invariably, associated with mitral or aortic lesions, either separately or combined. Their effects upon the heart are similar in kind to those of lesions seated in the left side, the points of departure for enlargement being the right auricle in cases of tricuspid obstruction or regurgitation, and the right ventricle in cases of pulmonic contraction or insufficiency.

[1] *Vide* Catalogue.

Examples of great enlargement of the right ventricle are observed in connection with congenital contraction of the pulmonary artery. Tricuspid regurgitation occurs not unfrequently without, strictly speaking, valvular lesions at this orifice. In certain cases of dilatation of the right ventricle, the auricular orifice becomes enlarged, the tricuspid valve not undergoing a corresponding increase in size. The consequence is insufficiency of the valve, or more or less patency of the orifice. Tricuspid regurgitation, under these circumstances, plays an important part in the production of certain pathological effects and symptoms of cardiac disease, viz., jugular turgescence and pulsation, general dropsy, etc., which will be presently noticed. In post-mortem examinations, valvular insufficiency from this cause is liable to be overlooked unless attention be directed specially to the size of the orifice, which, in its normal condition, should not greatly exceed four inches in circumference. It was remarked first by John Hunter, in his treatise on the blood, that the tricuspid valve is not so well adapted to afford complete protection to the auricular orifice as the mitral valve, and hence he infers that it is less important for this orifice to be protected on the right than on the left side. Mr. Adams,[1] of Dublin, and more recently and elaborately, Dr. T. W. King,[2] of London, have advocated the opinion that the tricuspid valve is disposed with special reference to regurgitation, and that an important part of its function is to permit a retrograde current through the auricular orifice when the right ventricle becomes over-distended. Dr. King bases his view of this "safety-valve function," as he terms it, upon the connection of the free extremities of the anterior and right curtains of the valve with the anterior wall of the ventricle, by means of the papillary muscles and tendinous cords. This connection he supposes to be such as to involve a separation of these two curtains from the remaining or posterior curtain when the accumulation of blood within the ventricular cavity is sufficient to over-distend the ventricle and carry far outward the anterior wall. An examination of a large number of hearts with reference to this point leads me to doubt whether over-distension of the ventricle produces the effect on the anterior and right curtains of the valve, at least in the majority of cases, which is attributed to it by Dr. King. The arrangement of the valve, however, is such that, when the ventricle

[1] Dublin Hospital Reports, vol. iv.

[2] Essay on "The Safety-valve Function in the Right Ventricle of the Human Heart," by T. W. King, Guy's Hospital Reports, vol. ii.

is distended, it is highly probable more or less regurgitation takes place at the commencement of the ventricular systole. But the different segments of the valve must quickly be brought into apposition by the systolic contraction of the ventricle, and further regurgitation prevented, provided the valve be sound and the auricular orifice not enlarged. It is reasonable to presume, with Hunter, that the difference in arrangement between the tricuspid and mitral valves is not without some important object; and it is not improbable that the object is to permit a certain amount of regurgitation. The fact that the right auricular orifice is somewhat larger than the left favors this supposition.[1]

Obstruction of the coronary arteries is to be included among the pathological effects of valvular lesions on the heart. Obstruction arises from encroachment of masses of fibrinous or calcareous deposit upon the mouths of these arteries, or an extension of deposit into the vessels themselves. The supply of arterial blood to the substance of the heart is diminished in proportion as the calibre of the vessels or their openings into the aorta are contracted. It is reasonable to infer that enfeebled muscular action must be the immediate result. It is not improbable that impaired nutrition, involving softening and leading to dilatation, may follow. According to the observations of Dr. Swain,[2] obstruction of the coronary arteries is found in a pretty large proportion of the cases of fatty degeneration. He observed this complication in twenty-five of eighty cases. The communications by anastomosis of the branches of the two coronary arteries not being very free, it has been supposed that obstruction of one may be sufficient to give rise to important pathological effects. Atrophy of the muscular substance of the heart has been observed in connection with extreme obstruction from the deposit of atheroma or calcareous deposit within these arteries. Formerly the paroxysms of severe pain which occur during the progress of some cases of organic disease of the heart, constituting the superadded affection known as *angina pectoris*, were attributed to coronary obstruction; but clinical observation has abundantly shown this association to be by no means constant.

As symptoms referable directly to the heart may be noticed, pain,

[1] The discussion of this subject belongs to physiology. The reader will find some remarks advocating the view here taken in the appendix to Hope on *Diseases of the Heart*, Am. ed., p. 546.

[2] Med.-Chir. Trans., *vide* Bellingham, op. cit.

palpitation, abnormal changes of the pulse, turgescence of the veins, and venous pulsation.

Pain.—Exclusive of paroxysms of suffering, frequently extremely severe, constituting the affection superadded to certain cases of organic disease of the heart known as *angina pectoris* (an affection to be considered hereafter), pain is not a prominent symptom of valvular lesions. In the majority of cases, this symptom is, in fact, wanting. Absence of pain is the rule, but occasionally patients complain of painful sensations, referred to the præcordia. A sense of constriction, uneasiness, or undefinable distress, is oftener met with than actual pain. These sensations are not distinctive of organic disease; they are quite as likely to occur in connection with merely functional disorder of the heart. When present, they do not indicate the particular seat of the lesions, but it is undoubtedly true that they occur more rarely in connection with mitral than with aortic lesions. This statement will be found to hold good equally with respect to paroxysms of angina pectoris. When the existence of valvular lesions is determined, by means of physical signs presently to be considered, the absence of pain, and, it may be added, tenderness or soreness of the præcordia, is of some weight in determining that the affection of the valves does not involve existing inflammation or endocarditis. But, as will be seen hereafter, pain, tenderness, or soreness are by no means always present when endocardial inflammation exists, so that absence of these symptoms is not proof against the existence of endocarditis.

Palpitation.—A person in health is not conscious of the action of the heart except when it is excited by exercise, mental emotion, or some other transient cause. The abnormal power or violence of this action in some cases of organic disease, however, renders it perceptible to the patient. It may force itself on his attention, and occasion annoyance or suffering, the action being, in some instances, simply more or less intense, and in others, at the same time, irregular or intermitting. The consciousness of an undue force of impulsion is by no means a constant symptom of valvular affections. It does not occur till the heart becomes enlarged as a result of these affections. It is, in fact, due, not directly to the lesions of the valves or orifices, but to the hypertrophy to which they give rise. When the patient complains of the beating of the heart, the impulse is found, on applying the hand over the præcordia, to be

abnormally forcible. Portions of the dress or body are visibly moved by the violence of the action. But the patient often does not complain of this symptom, and may not appear even to notice it, when to the observer it is strongly marked. It is not unusual for patients to say that they have never experienced palpitation, when the action of the heart is perceived by the hand, applied over the præcordia, to be extremely violent and irregular. The explanation of this is, the abnormal force having been developed gradually and imperceptibly, the mind has become habituated to it, and is unconscious of it, at least unless the attention be directed to it. Palpitation, therefore, may be present as an objective when it is wanting as a subjective symptom. Hence, also, the inconvenience which it occasions does not always correspond with the degree in which it actually exists. Other things being equal, the increased violence of the heart's action is proportionate to the amount of hypertrophy, and especially hypertrophy of the left ventricle.

The consciousness of undue violence or of irregularity of the heart's action, exclusive of other circumstances, is not significant of organic disease. On the contrary, if the patient complain of this as a prominent symptom, the presumption is that organic disease does not exist. Palpitation from functional disorder always occasions great uneasiness, and generally intense anxiety and alarm. It is quite otherwise with palpitation incident to organic disease. It is surprising how insensible patients frequently are to the excessive force and great irregularity of the action of the heart, when due to structural affections, and how indifferent they often are when conscious of palpitation. The contrast, in this respect, between cases of organic disease and those of merely functional disorder is very striking. Other points serve to distinguish functional palpitation from that due to organic disease. The latter is less violent, but, to a greater or less extent, constant, while the former occurs in paroxysms, in the intervals of which the heart is tranquil. Palpitation from organic disease, increased beyond its habitual amount, is occasioned generally by some obvious cause, and more especially by muscular exercise. Functional palpitation occurs often when it cannot be traced to any exciting cause, and is more likely to occur when the patient is at rest than when engaged in active exertion. The former takes place more frequently in the daytime, the latter during the night. These and other points pertaining to the differ-

ential diagnosis will be considered more fully in treating of functional disorders of the heart.

As regards the relations of palpitation to the different valvular lesions, undue violence of the heart's action occurs more frequently, and, as a rule, is more marked in cases of aortic than in cases of mitral lesions. This is owing to the fact that the former, more than the latter, tend to give rise to hypertrophy of the left ventricle. The violence of the impulse, of course, depends on the amount of enlargement by hypertrophy rather than by dilatation, and on the activity of the muscular contractions. The patient is more likely to perceive and suffer from the violence of the heart's action if the hypertrophy has been developed rapidly than if its progress has been very gradual. The irregularity, of which the patient may or may not be conscious, depends, in a great measure at least, on the variations in the quantity of blood delivered to the cavities, in consequence of the interruption to the currents by the obstructive or regurgitant lesions. Irregularity of action due to these causes will be considered in connection with the pulse.

Pulse.—The characters of the pulse are often of considerable assistance in determining the situation, nature, and extent of lesions affecting the valves and orifices of the heart. The abnormal variations, which are important to be considered with reference to the points just mentioned, are its size and strength as compared with the heart's impulse, its rhythm, the equality or inequality of successive beats, its quickness or slowness, the duration of the movement of the artery under the finger, etc. The frequency of the pulse, although important as representing the general condition of the circulation and the state of the vital forces, has no special significance as regards valvular disease.

In mitral lesions attended by regurgitation, the size and strength of the pulse are diminished in proportion to the quantity of blood driven backwards, by the systolic contraction of the left ventricle, into the left auricle. If a small quantity only regurgitates, the pulse may still retain considerable volume and force; but if the amount of regurgitation be large, the pulse is notably weak and small. The weakness and smallness of the pulse are in contrast with the impulse of the heart, as felt by the hand applied over the præcordia, provided the left ventricle be hypertrophied and the action of the heart vigorous. The pulse may be regular, but often, in an advanced stage of the affection, its rhythm is disturbed; it

becomes irregular or intermitting. This, however, is due, not directly to the regurgitation, but to the condition of the muscular walls of the organ. Inequality of the pulse, that is, variation of successive beats as respects size, force, etc., is less characteristic of mitral regurgitant lesions than of those attended by obstruction. The pulse in cases of mitral regurgitation is not unfrequently quick or vibratory. It presents this quality when the regurgitation is not excessive, and the left ventricle is moderately hypertrophied. The frequency here, as in the other varieties of valvular lesions, depends on the vital condition of the heart.

Mitral obstructive lesions equally, but in a different manner, involve diminution of the size and strength of the pulse. In regurgitation, the pulse is rendered small and weak by the deduction of the blood which regurgitates from the quantity which would otherwise be propelled into the aorta with each systole. In obstruction, the blood not passing from the left auricle with sufficient freedom, the left ventricle fails to receive the quantity which should be propelled into the aorta. In the one case, the left ventricle is abundantly supplied, but it is not capable of conveying all its contents into the arterial system in consequence of the insufficiency of the mitral valve; in the other case, the supply to the left ventricle is deficient, and the blood accumulates in the left auricle and pulmonary vessels. In both cases the effect is the same so far as regards the lessened quantity of blood propelled into the aorta, and hence in both alike there occurs abnormal diminution of the size and strength of the pulse. The pulse, under these circumstances, as in cases of mitral regurgitation, is sometimes quick, vibratory, or jerking. Mitral contraction, when extreme, renders the pulse not only small and weak, but often irregular, intermitting, and unequal. The latter variations are observed especially when the pulse is at the same time frequent. The inequality depends on the varying quantity of blood which passes from the auricle to the ventricle between the successive systolic contractions of the ventricles. When the orifice is much obstructed, various circumstances prevent an equal supply of blood to the ventricle prior to the ventricular systoles, and in proportion to the quantity of blood propelled into the aorta, other things being equal, will be the size and force of the pulse. When, from any cause, the supply of blood preceding the ventricular systole is less than usual, the pulse, which represents the systole of the left ventricle, is unusually small and weak. Under these circumstances, the action of the heart is often

increased in an irregular manner. A greater deficiency of blood than usual causes the ventricle to contract for several beats with more frequency, as if to compensate by the number of systolic movements for the deficient supply of blood; hence, it is not unusual after several regular beats of the pulse, having a certain volume and strength, for a series of rapid beats to ensue, which are notably small and weak. As remarked by Dr. Adams, "it appears as if there were two pulses, one slow and deliberate for two or three beats, succeeded by three or four rapid and indistinct pulsations."[1] Intermittency of the pulse may represent intermittency of the heart's action, but it is sometimes observed when there is not a corresponding interruption in the heart's impulse. The apex-beats may be felt by the hand over the præcordia to take place in regular succession, while the pulse is found to intermit more or less frequently. This occurs in some cases of mitral obstruction, the explanation being that the quantity of blood delivered from the auricle to the ventricle, through the contracted orifice, is at times insufficient for the wave through the remote arteries to be perceived by the touch. The beats which, under these circumstances, are lost at the radial artery, may be distinguishable at an artery larger in size and nearer the heart, viz., the carotid. Intermittency of the pulse, it must be recollected, is a peculiarity of the circulation in some persons in health. An intermission, or the loss of a beat, occurs more or less frequently, the person not being conscious of its occurrence. It is not, therefore, intrinsically a symptom of disease. It is a curious fact that in persons who present this idiosyncrasy the pulse ceases to be intermittent in disease attended by febrile movement. A reappearance of the intermissions, under these circumstances, is evidence of the return of health. Weakness, smallness, and irregularity, as well as intermittency and even inequality, it is to be borne in mind, are not distinctive of mitral or other valvular lesions. All these characters of the pulse may occur in cases of enlargement or fatty degeneration uncomplicated with lesions of the valves or orifices. All may occur, moreover, in merely functional disorder of the heart. A distinguishing point pertaining to the latter is, that they occur only during paroxysms of palpitation presenting the distinctive features of palpitation from nervous disorder, while, occurring in connection with valvular lesions, they are either constant or frequently recurring, and un-

[1] Dublin Hospital Reports, vol. iv. From Bellingham, op. cit.

attended by the features which distinguish functional palpitation. It is also to be borne in mind that in cases of mitral obstruction, provided the contraction of the orifice be not extremely great, the pulse may present sufficient size and strength, and it may be regular and equal. It becomes irregular, intermittent, and unequal more especially at an advanced stage of disease, when great dilatation and weakness of the muscular walls are superadded to the valvular affection. It follows from these remarks that the diagnostic value of the abnormal variations of the pulse which have been mentioned, depends not on the characters intrinsically, but, in a great measure, on the associated circumstances. Considered alone, their import is not distinctive of the nature, situation, or existence of organic disease, but taken in connection with other symptoms, and with physical signs, they often supply important information. Thus, the existence of mitral lesions having been ascertained by means of signs which are sufficiently distinctive for the diagnosis, the characters of the pulse which have been noticed will assist in determining whether the lesions are obstructive or regurgitant; but they afford more aid in estimating the extent to which either regurgitation or obstruction interferes with the cardiac circulation. As regards the differential diagnosis between regurgitation and obstruction, the pulse is more likely to be unequal, irregular, and intermitting in the latter than in the former, irrespective of that degree of dilatation and weakness of the heart which may induce these characters with or without valvular lesions. The amount of obstruction or regurgitation is represented by the smallness and weakness of the pulse, the more these characters are in contrast with the strength of the heart's impulse or felt by the hand in the præcordia. It will be recollected that mitral obstructive and regurgitant lesions are not unfrequently combined. The symptomatic phenomena referable to the pulse will, of course, be more marked in such cases.

Aortic lesions giving rise to obstruction are not characterized by a pulse weakened in proportion to the diminished quantity of blood propelled from the left ventricle. Even when the contraction of the orifice is great, the pulse generally retains considerable force. In an instance in which the orifice was reduced to the size of a quill, the pulse was neither small nor weak in a marked degree. This fact, which at first view may seem inconsistent, is intelligible when it is considered that a primary effect of aortic obstruction is hypertrophy of the left ventricle. The increased muscular power of this ventricle thus in a measure compensates for the reduction

in size of the aortic orifice.[1] Moreover, the arteries are not unfilled to the same extent as in cases of great mitral obstruction and regurgitation, and the momentum communicated by the hypertrophied ventricle to the column of blood contained in the arteries may be sufficient to produce a pretty strong pulsation of the arterial trunks, even when obstructive aortic lesions exist to a considerable extent. Nevertheless, in cases of extreme contraction (to which reference has been made), in which the orifice has been found scarcely to admit a small probe, the obstruction is too great to admit of compensation, and the pulse, under these circumstances, is small and weak. In cases of aortic obstruction, when enlargement of the heart has ensued, and especially when the muscular power of the organ is much diminished, the pulse may become irregular, intermitting, and unequal. These deviations occur alike in aortic and mitral lesions at an advanced period of the disease. They occur, however, less frequently, at a later period, and in a less marked degree, in cases of aortic obstruction than in cases of either mitral obstruction or regurgitation. Irregularity and inequality are thus, in some measure, diagnostic of lesions affecting the mitral orifice as contrasted with those affecting the aortic orifice; but it is to be borne in mind that they occur in cases of dilatation, fatty degeneration, etc., uncomplicated with any affection of the valves or orifices. When the amount of aortic obstruction is sufficient to affect, in a marked degree, the size and force of the pulse, the impulse felt in the præcordia may be abnormally strong, owing to hypertrophy of the left ventricle. It is especially in cases of this description that a marked contrast between the pulse and the heart's impulse is observed.

Aortic lesions giving rise to regurgitation, if the regurgitant current be considerable, are characterized by a pulse which is in some measure diagnostic. When the aortic valves are sufficient, the column of blood contained in the arteries is supported by them, after the systolic contraction of the ventricle, and the elastic recoil of the arterial coats contributes in propelling the blood in its out-

[1] Dr. Blakiston conjectures that an additional mode of compensation consists in a prolongation of the systolic contraction when considerable aortic contraction exists, the blood (quoting his language) being "gradually squeezed through the contracted orifice." (*Practical Observations on certain Diseases of the Chest, etc.*, Am. ed., p. 225.) The reader will find cited, in connection with this subject, in that work, several cases illustrating extreme aortic contraction, in which the symptoms of cardiac disease were very slightly manifested.

ward current. But if the valves are insufficient, the column of blood being incompletely supported after the ventricular systole, a quantity, greater or less, according to the extent of the insufficiency of the valves, flows backwards into the ventricle, and the recoil of the arterial coats acts alike in producing an onward and a regurgitant current, so that when the contraction of the left ventricle takes place, the blood propelled into the aorta meets a regurgitant instead of an onward current. Clinical observation shows that under these circumstances, as first pointed out by Dr. Corrigan, of Dublin, the pulse is notably quick and short, that is to say *jerking*. The artery strikes the finger suddenly and often with considerable force, and appears instantly to recede. This has also been called a "collapsing pulse." It occurs, as a rule, in cases of aortic lesions with considerable insufficiency, and the jerking or collapsing feature is usually strongly marked. It is not, however, a symptom so distinctive of aortic regurgitation, as it appears to be considered by Dr. Corrigan and others. Clinical observation shows that the pulse is sometimes jerking in cases of mitral obstruction and regurgitation, the aortic valves being unaffected. But it is undoubtedly true that the symptom is much oftener present and more strongly marked in cases of aortic regurgitation. It is not a symptom of aortic obstruction, and inasmuch as the physical signs enable the diagnostician to determine the existence of lesions affecting the valves or orifice of the aorta, it is a symptom of importance as indicating that the lesions here situated, are of a kind to permit regurgitation. Taken alone, it is certainly not reliable as evidence of the presence of aortic lesions, but these having been ascertained, it aids in discriminating between obstructive and regurgitant lesions, or rather it indicates the existence of the latter either with or without the former.

Visible pulsation of arterial trunks superficially situated, such as the subclavian, carotid, temporal, brachial, radial, etc., is a symptom somewhat characteristic of aortic regurgitation. If the insufficiency of the valve be great, an effect of the collision of the retrograde diastolic current, and the onward systolic current in the vessels, is to cause the latter, as it were, to be "suddenly thrown from their bed, bounding up under the skin." The visible pulsation is due not alone to the diastolic movements of the coats of the vessels, but to the locomotion of the arteries. They "sometimes appear like worms under the skin, wriggling into tortuous lines at each pulse."[1] The connection of this symptom with aortic re-

[1] Dr. Williams. Bellingham, op. cit.

gurgitation, was first pointed out by Dr. Corrigan.[1] It is by no means a symptom which belongs exclusively in this connection. It is observed in a marked degree, not unfrequently, in thin persons when the arteries have become rigid or calcareous, with or without cardiac disease. In a moderate degree it is sufficiently common in various pathological associations. But it is a symptom usually present in cases in which considerable regurgitation takes place, and is generally strongly marked. Dr. Walshe states that no well-marked case of aortic regurgitation has ever fallen under his notice, in which visibleness in the superficial pulses was not more or less present, and that he has never observed highly-marked and extensive visible pulsation without aortic regurgitant disease.[2] The coexistence of considerable mitral obstruction with regurgitation, does not, as supposed by Hope, prevent this symptom from being present, at least in all cases. Taken in conjunction with the signs which establish the diagnosis of aortic lesions, it concurs with the jerking pulse, in signifying that the lesions are of a nature to render the valve insufficient.

Another symptom pertaining to the pulse, has been pointed out by Dr. Henderson, as significant of aortic regurgitation, viz: prolongation of the interval between the pulsation of the radial artery and the heart's impulse.[3] The interval, according to Dr. Henderson, is sometimes so much lengthened "that the heart and the radial artery seem to beat with a distinct alternation." The rationale of this symptom is intelligible, in view of the conflicting currents within the large vessels which have been referred to in connection with the production of a jerking pulse, and visible pulsation of the arteries. Some observers have been led to doubt the frequent occurrence of this symptom. That it characterizes certain cases in which the regurgitation is excessive, is not to be denied. In a case of mitral and aortic regurgitation, with great hypertrophic enlargement, which came under my observation, the interval between the apex-beat and the beat of the radial artery, was longer than that between the first and second sounds of the heart. The radial pulse was, in fact, in much closer relation to the diastole than to the systole. The interval between the apex-beat and the pulsation of the carotid artery, was less, being about the same as exists normally between the apex-beat and the radial pulse. The visible movements

[1] Edinb. Med. and Surg. Journ., vol. xxvii. 1832.

[2] Op. cit., English second edition, p. 265.

[3] Edinburgh Monthly Journal, May, 1843.

of the arteries, and the jerking character of the pulse were strongly marked in this case.[1] The *sphygmoscope*, recently devised by Dr. Scott Alison, of London, is admirably adapted to illustrate the relative occurrence of the apex-beat and the arterial pulse, in different situations.

Turgescence of the Veins and Venous Pulsation.—Abnormal fulness of the veins occurs whenever an obstacle exists to the free entrance of blood into the right auricle. An obstacle exists, when, from any cause, the right auricle is already full, or distended with blood. Various abnormal conditions involve this result. The most direct and efficient causative condition is contraction of the tricuspid orifice. But this is exceedingly rare; so much so, that the probabilities of its existence in a given case of distension of the right auricle, are hardly sufficient for it to be taken into account. Tricuspid regurgitation is another condition leading directly and efficiently to the result. This, although probably not of very frequent occurrence, at least in an abnormal degree, exists much oftener than tricuspid contraction. The explanation of distension of the right auricle, when, from insufficiency of the tricuspid valve, a portion of the contents of the right ventricle is driven backward with each systole, is obvious. Dilatation of the auricle follows sooner or later. Lesions affecting the valve or orifice of the pulmonary artery also induce distension and dilatation of the right auricle, exerting an effect primarily on the right ventricle. Contraction and insufficiency here situated, have been seen to be as infrequent as tricuspid obstruction, excepting instances of congenital lesions. In the affections developed after birth, therefore, these are to be excluded as probable conditions giving rise to venous turgescence. Distension and dilatation of the auricle, however, occur irrespective of lesions affecting either the tricuspid or pulmonic orifice. They occur, as has been seen, in connection with mitral and aortic lesions which involve either obstruction or regurgitation, separately or combined. The right ventricle in these cases first becomes overdistended and enlarged, and, consecutively, distension and dilatation of the right auricle follow. Turgescence of the superficial veins is therefore observed, not alone in cases of tricuspid and pulmonic, but also in cases of mitral and aortic lesions when the latter have led to over-repletion and enlargement of the right side of the heart. It is equally a symptom of the latter when not in-

[1] Case of Hart, Private Records, vol. x. p. 586.

duced by valvular lesions. Obstruction to the pulmonary circulation from any cause, for example, from emphysema of the lungs, occasions an undue accumulation of blood within the right ventricle and auricle, leading perhaps to enlargement, and a consequent obstacle to the free escape of blood from the systemic veins. Finally, pressure on the vena cava by an intra-thoracic tumor produces obstruction and venous turgescence. Thus, marked fulness of the veins of the head and neck is observed in some cases of aneurism of the arch of the aorta. As a symptom, then, this is not distinctive in itself of cardiac disease, nor when it proceeds from the latter, does it point to the seat, or even denote the existence of lesions of the valves or orifices. Exclusive of the cases in which it is an immediate effect of extra-cardiac obstruction, it simply shows that the right auricle is either dilated or over-distended.

Venous turgescence may be apparent whenever the superficial veins are visible, but it is usually most conspicuous on the neck, in the jugulars, and the venous branches communicating with them. In some instances of extreme turgescence the vessels are developed so as to present a varicose appearance. These veins may be habitually full and dilated, as they are seen to be temporarily during prolonged expiratory efforts in singing, playing on wind instruments, straining, and in paroxysms of spasmodic cough. If the cardiac obstruction be considerable, when pressure is made on a vein high on the neck, the vessel remains distended below the point of pressure, and may be refilled after the contents of the vessel have been pressed backward by the finger, showing not only a resistance to the gravitation of the blood, but a reflux current.

Although, intrinsically, this symptom appears wanting in precise diagnostic significance, taken in connection with the physical signs which establish the nature and seat of organic lesions of the heart, it possesses considerable value. If it proceed from lesions situated at the tricuspid or pulmonic orifices, the concurrent signs will show the existence of these lesions, and the degree of venous turgescence will be, to some extent, an index of the extent to which they occasion immediate obstruction. If, on the other hand, it proceed (as it does in the vast majority of cases) from aortic or mitral lesions, the signs enable us to localize these, and the turgescence is then evidence, and its degree in some measure a criterion, of the effect which they have produced on the right side of the heart. It shows that the mitral or aortic lesions involve an amount of obstruction which will be likely to lead to enlargement of the right auricle and

ventricle, if it have not already taken place. It shows also an effect on the systemic circulation which involves a liability to other effects dependent on it, viz., dropsy, extravasation of blood, and hemorrhage. In these points of view it is a symptom which deserves attention in the examination of patients affected with cardiac disease. It is, of course, understood that the value of this symptom, in its relation to disease of the heart, depends on the absence of extra-cardiac conditions, such as emphysema, aneurism, &c., which may give rise to it. Careful examination will generally enable the diagnostician either to exclude these conditions or to ascertain their presence in individual cases.

Venous pulsation is a diastolic movement of the veins, visible, and sometimes even appreciable by the touch, occurring, in general, synchronously with the ventricular systole of the heart. The movement is due to a retrograde current or impulse communicated to the blood contained in the veins by the contraction of the right ventricle. It is to be distinguished from the movements occasioned by respiration, with which every one is familiar, and also from those communicated by subjacent arteries. With the latter it is liable to be confounded, unless care be taken to avoid it. It may be avoided by pressing the blood from the pulsating vein, and ascertaining whether the pulsation continues when the vessel is empty; or, if practicable, by stopping the pulsation in the artery by pressure, and observing if the venous pulsation continues. The movements due to respiration may be arrested by causing the patient to suspend breathing for a few seconds. Pulsation is rarely observed elsewhere than in the veins of the neck. It is often limited to the jugular veins just above the clavicles. It may be limited to one side of the neck, and, when this is the case, it is usually observed on the right side. In some instances, however, the pulsation extends to the superficial veins at remote parts of the body. It has been observed even on the dorsal surface of the hands.[1] Pulsation is usually accompanied by turgescence of the veins, and is especially marked at the end of the act of expiration, when the fulness is greatest. It may also be increased by pressure on the vein above the point where it is observed. It varies in degree or force between a very gentle undulatory and frequently intermittent movement perceptible to the eye, and a movement

[1] A Case of Pulsation in the Veins of the Upper Extremities. By Charles Benson, M. D. *Dublin Journal of Medical Science*, vol. viii., series No. 1, 1836. *Vide* Stokes, op. cit., Am. ed., p. 219.

which is not only seen, but communicates a sensation to the finger sufficiently distinct but never strong.

As a symptom of cardiac disease, venous pulsation was first described by an Italian author, Lancisi, who ascribed it to dilatation of the right ventricle. The phenomenon, however, had been previously noticed by another Italian writer, Testa.[1] The inquiry which first arises is, Does it invariably denote disease? Mr. J. W. King, in connection with the subject of the safety-valve function of the tricuspid valve, adduces cases to show that it occurs independently of any organic disease of the heart. And Dr. Francis Sibson states that "a slight systolic pulsation is visible, below the sterno-cleido, in the superficial jugular, in thirty-nine persons out of forty, when they lie down."[2] Assuming that a venous pulsation may exist without being abnormal, the supposition which has been entertained, that it may be produced by the impulse communicated by the contraction of the left ventricle being transmitted through the capillary vessels (*vis à tergo*), is not tenable. It must be due, in health as in disease, to a reflux current, and hence it follows, assuming its occurrence to be synchronous with the ventricular systole, either that an amount of tricuspid regurgitation, irrespective of disease, sometimes occurs sufficient to give rise to a retrograde current extending to the cervical veins, or, as contended by Hope, the expansion of the curtains of the tricuspid valve during the ventricular systole suffices to impart to the blood contained in the right auricle a momentum which extends to the column of blood contained within the veins for a certain distance. The latter view is not improbable, and both explanations are perhaps admissible. But it is only when the pulsation is slight and circumscribed that there is room for doubt as to its being a symptom of disease. If it be marked or extensive, it is to be considered, generally, as evidence of tricuspid regurgitation. Its significance, under these circumstances, renders it a valuable diagnostic symptom. It is more valuable in a positive than in a negative point of view: that is, while its presence in a marked degree is, in general, evidence of tricuspid regurgitation, its absence is not proof of the non-occurrence of this regurgitation. An important element in its production is involved, in addition to insufficiency of the tricuspid valve, viz., increased, or at least undiminished, muscular power of the right

[1] *Vide* Stokes, op. cit., Am. ed., p. 214, note.

[2] Medical Anatomy. London. Fasciculus, No. 1.

ventricle. If the contraction of this ventricle be feeble, from dilatation or weakness, the regurgitant current is not strong enough to extend much, if at all, beyond the auricle into the veins. On the other hand, the force of the regurgitation and the reflux into the veins, other things being equal, will be commensurate with the power with which the right ventricle contracts. Hence, it is obvious that the symptom under consideration is not only valuable as evidence of tricuspid regurgitation, but also as constituting, in some measure, an index of the energy of the systole of the right ventricle. The conditions most favorable for the production of the venous pulse are free tricuspid regurgitation and hypertrophy of the right ventricle. These combined conditions are present in connection not only with valvular lesions confined to the right side of the heart, but, as has been seen, with lesions affecting the aortic and mitral valves and orifices, in a certain proportion of cases. It is easy to understand that hypertrophy of the right ventricle, without tricuspid regurgitation, may exaggerate the pulsatory movements of the cervical veins which are often observed in a slight degree in health. But it is probably correct to say that hypertrophy of the right ventricle alone does not give rise to this symptom in a marked degree, and, therefore, that regurgitation is to be inferred in such cases.

Jugular pulsation is to be explained in the manner just stated in most of the instances in which it is observed. In a certain proportion of instances, however, this symptom is otherwise produced. The systolic contraction of the right auricle may cause a movement of the blood in a retrograde direction sufficiently to give rise to venous pulsation. Experimental observations show that the auricular systole precedes, by a very brief interval, the ventricular, the former being, as it were, continued into the latter. Venous pulsation due to auricular contraction should therefore precede slightly the arterial pulse or apex-beat of the heart, while clinical observation shows that generally the venous pulse lags a little behind that of the arteries, the reflux venous current being somewhat slower than the direct arterial current. The point just stated suffices for the discrimination between an auricular and ventricular venous pulse, and it is perhaps true that adequate attention has not been given to this discrimination in observations of disease. But it is probably correct to consider venous pulsation as referable, in the vast majority of cases, to the action of the right ventricle. In a case recently under observation, a double undulation of the super-

ficial jugular vein on the right side of the neck existed, one preceding and the other coinciding with the ventricular systole.[1] The veins of the neck were extremely turgid; and examination after death disclosed lesions of the tricuspid orifice involving considerable obstruction as well as regurgitation, together with great dilatation of the right auricle, and enlargement, by hypertrophy, of the right ventricle.

Symptoms and Pathological Effects referable to the Circulation.

The symptoms just considered, viz., the abnormal variations of the pulse, turgescence of the veins, and venous pulsation, relate to the circulation, but they are due immediately to the heart, and hence represent directly its morbid conditions. Other results of disease relate to the circulation, but are developed as ulterior effects of valvular affections, involving intermediate conditions, and, therefore, representing indirectly those which are seated in the heart. Under the present head are embraced two important events incidental to the clinical history of affections of the valves and orifices, to wit, dropsy and arterial obstruction from masses of solid deposit detached from the endocardial membrane, and carried with the current of blood into the vessels. Important pathological events in addition to these relate to the circulation, but will be more appropriately considered in connection with the phenomena referable to different organs. Such are hemorrhage, apoplectic extravasation, flux, etc.

Cardiac Dropsy.—Serous transudation into the areolar tissue beneath the integument, into the pulmonary parenchyma, the peritoneal and other serous cavities, in other words, general dropsy, occurs sooner or later in a large proportion of the cases of valvular affections in which a fatal termination does not take place in consequence of some intercurrent or incidental disease. General dropsy

[1] Hospital Records, New Orleans Charity Hospital, vol. xiv. p. 271. The relation of pulsation of the cervical veins to the contractions of the right auricle is an interesting subject for further clinical study than appears to have been as yet given to it. Prof. Skoda attributes this symptom, in certain cases, to the auricular systole. *Vide* On the Functions of the Auricles of the Heart, translated from Schmidt's *Jahrbücher*, July, 1853, by W. O. Markham, M. D., *Brit. and For. Med.-Chir. Rev.*, Feb. 1854.

dependent on disease of the heart is called cardiac dropsy. It may be due to other pathological conditions, generally to disease of the kidney, when it is distinguished as renal dropsy. It appears first, as a rule, in the form of œdema of the feet and ankles, which gradually extends over the lower extremities. Œdema of the face follows, sometimes occurring nearly simultaneously with, and occasionally prior to, swelling of the feet. It may extend over the whole body, constituting anasarca. The lower extremities, in some cases, become enormously swollen. Erythema, and occasionally gangrene, result from the extreme distension of the integument. Blisters, ulcerations, and cracks are other consequences, giving exit to the transuded liquid, which flows away in abundance. The surface, more especially the face, presenting at the same time more or less venous congestion, the general aspect in cardiac dropsy is somewhat characteristic. The face has a dark or dusky hue, forming a striking contrast to the pallid complexion which is usually marked in cases of dropsy from renal disease, or when it occurs in connection with anæmia from whatever cause induced. Effusion into the different serous cavities takes place subsequently to the subcutaneous œdema. Dr. Walshe ranks œdema of the pulmonary parenchyma over dropsy of the cavities, as respects frequency of occurrence. Mention will be made of this under the head of pathological events referable to the pulmonary system. The different serous cavities are by no means equally liable to dropsical effusion. The following is an enumeration of the several cavities after the relative frequency with which they are found to be affected in diseases of the heart: Peritoneal, pleural, pericardial, arachnoid, subarachnoid, and the tunica vaginalis.[1] More or less of these, and sometimes all of them, present dropsical accumulation in the same case.

The occurrence of dropsy has reference to the situation, nature, and degree of valvular lesions. Tricuspid contraction is the lesion which most directly and efficiently tends to give rise to this effect; but this lesion is exceedingly infrequent. Tricuspid regurgitation exerts a similar tendency, but in a less degree. Dropsy does not uniformly occur in the cases in which turgescence of the veins and venous pulsation show marked insufficiency of the tricuspid valve. Of lesions situated in the left side of the heart, mitral contraction is most likely to give rise to dropsy. Mitral regurgitant lesions

[1] Walshe, Diseases of the Lungs and Heart, English edition, p. 648.

come next in order, as regards this tendency. Aortic lesions, oftener than mitral, continue and terminate without this pathological effect; but they are by no means always exempt from it. By what mechanism is the dropsy produced? So far as concerns the agency of the cardiac lesions, the intermediate morbid condition is passive congestion of the systemic veins. To understand the *modus operandi*, we have to inquire in what manner the different obstructive and regurgitant lesions induce this venous congestion. This is sufficiently intelligible as regards lesions seated at the tricuspid orifice. The relations of the systemic veins to the right side of the heart are such that this consequence of obstruction and regurgitation situated here is at once evident. It is not less clear when the lesions are situated at the mitral and aortic orifices, in view of the effects of these on the right side of the heart, which have been already considered. Dropsy follows the latter lesions in consequence of the distension and dilatation of the right ventricle and auricle to which these lesions give rise. Repletion of these cavities constitutes an obstruction which may induce sufficient congestion of the systemic veins to lead to serous transudation. Thus, so far as concerns the relation of dropsy to valvular lesions, they all alike produce this effect by obstruction at the termination of the systemic venous system, viz., the right auricle; tricuspid lesions, involving directly this obstruction, and lesions situated at the mitral or aortic orifice, leading indirectly to the same result. Dr. Blakiston supposes insufficiency of the tricuspid valve to exist whenever dropsy proceeds from lesions situated in the left side of the heart. It has been seen that the enlargement of the right ventricle, consequent more especially on mitral lesions, not unfrequently induces an augmentation of the tricuspid orifice, rendering the valve insufficient. The regurgitation which takes place in these cases doubtless adds considerably to the congestion of the systemic veins. Dr. Blakiston presents a collection of cases illustrative of this fact.[1] But clinical observation shows that dropsy may occur in connection with lesions situated in the left side of the heart without the intervention of either tricuspid contraction or abnormal insufficiency.

From the foregoing remarks it follows that the occurrence of dropsy, other things being equal, in cases of mitral or aortic lesions, will depend, not immediately on the nature and extent of these

[1] Practical Observations on Certain Diseases of the Chest, and on the Principles of Auscultation, Am. ed., 1848, p. 231 *et seq.*

lesions, but on conditions induced thereby, which relate to the right side of the heart. When to distension of the right ventricle and auricle is added either dilatation or weakness of the right ventricle, the venous obstruction due to over-accumulation of blood is increased by the diminished ability of this ventricle to contract and expel its contents. In point of fact, dilatation of the right ventricle or weakness from fatty degeneration, or other causes, precedes, in the great majority of cases, the occurrence of dropsy. Dropsy, therefore, is an event which usually belongs to an advanced period of organic disease, and it is frequently a precursor of a fatal termination. Enlargement of the right side of the heart, especially if accompanied by degeneration of structure or great muscular weakness, may induce dropsy when valvular lesions are not present. The occurrence of dropsy, thus, when aortic or mitral lesions are present, is evidence that the effects of these lesions on the right side of the heart, which have been considered under another head, have taken place.

The mechanism of dropsy occurring in connection with valvular affections, so far as at present considered, involves simply mechanical pressure. The serous or watery portion of the blood transudes through the coats of the vessels in consequence of their distension, in the same manner that œdema of an extremity is induced by the obliteration of an important venous trunk coming from it. Abnormal conditions in addition to those giving rise to venous obstruction, however, may concur in producing cardiac dropsy. The frequent concurrence of other causes is shown by the absence of dropsy in cases in which the conditions pertaining to the heart must necessarily have involved, for a long period, marked congestion of the systemic veins. Aortic and even mitral lesions, involving a great degree of regurgitation and contraction, may end after a protracted duration without having led to dropsy. It is shown also by the want of proportion, which all clinical observers have noticed, between the occurrence or the amount of dropsical transudation and the degree of obstruction which the cardiac conditions involve. Not only are these conditions marked in cases in which dropsy has not occurred, but dropsy occurs in other cases in which these conditions are comparatively slight. Evidently, then, something more than mechanical pressure is concerned in the production of dropsy in at least a certain proportion of the cases of cardiac disease in which this pathological effect takes place. Clinical experience shows that in some instances a concurring morbid con-

dition is disease of kidney. Disease of heart and the affection of kidney commonly known as Bright's disease, are occasionally found associated. The causes which induce dropsy in the latter affection, then co-operate with those relating to the heart. Renal and cardiac dropsy are, in fact, combined. Anæmia or hydræmia, arising from various causes, may determine the occurrence of dropsy, when the cardiac lesions of themselves would not have produced it. Transudation of the attenuated serum, it is well known, takes place as a result of this condition of the blood when neither cardiac nor renal disease exists. Dr. Walshe attaches importance to the impaired nutrition of the walls of the vessels from the strain incident to prolonged distension, as a subsidiary cause of transudation. These additional conditions are to be taken into account in explaining the production of dropsy in cases of cardiac disease; and they serve to explain the efficiency of therapeutical measures in some instances in which the cardiac lesions are such as to render the continuance of venous congestion inevitable.

Arterial obstruction by fibrinous deposits detached from the valves or orifices of the heart. Embolia.—This subject has only within the past few years engaged the attention of pathologists and clinical observers. A sufficient number of facts have been ascertained, to show that it is entitled to be considered among the interesting and important effects of valvular affections. Further investigation, however, is required, in order to determine fully how far it is involved in the pathological history of these affections. As already remarked, in describing the morbid appearances which are presented in cases of lesions of the valves and orifices, the deposits distinguished as vegetations, warty excrescences, etc., are frequently, in examinations after death, found to be so slightly adherent that it is reasonable to suppose the current of blood to be sufficient, in some instances, to detach them during life. They are then carried onward with the current in the course of the circulation, until they reach an arterial trunk smaller than their own dimensions. Here they are arrested, and, becoming wedged in the vessel, they act as plugs, obstructing the passage of blood in the artery and its branches beyond the point at which they are lodged. These detached deposits are called *emboli*, or migratory plugs, by Prof. R. Virchow, of Würzburg, who appears to have been the first to make obstruction of the systemic arteries, as thus produced, a subject of scientific

study.[1] Others have contributed the results of their researches, among whom Dr. William Senhouse Kirkes, of London, is to be especially mentioned.[2] The subject is designated by the term *embolia*. This term, however, embraces migratory plugs formed elsewhere than in the heart. They may be formed in the arteries and in the veins, in the latter case being sometimes transported to the heart, and thence into the branches of the pulmonary artery. Those which consist of vegetations or warty excrescences detached from the valves, do not embrace all which may be derived from the heart. Recently deposited fibrin and coagulable lymph occurring during the progress of endo-carditis, and fibrinous clots formed under various pathological circumstances, also constitute emboli. The subject, therefore, is not limited to valvular lesions in its application, and will be referred to hereafter, in treating of endo-carditis and the formation of coagula within the heart.

Detached deposits from within the heart, in cases of chronic valvular lesions, are derived, in the vast majority of cases, from the left ventricle, since lesions affecting the valves of the right side of the heart are exceedingly infrequent. The obstructed arteries, therefore, belong to the systemic class, the emboli passing with the current of blood into the aorta and along the successive arterial trunks, until arrested in their progress by branches, the calibre of which is too small to permit their farther progress. The situation in which an embolus becomes fixed, will depend on its size and the direction which it happens to take. Owing to the large quantity of blood sent to the brain, it will be likely to take that direction, and produce obstruction of some one of the cerebral arteries. Cases reported by Dr. Kirkes, and others, seem to show that in this manner, circumscribed softening of the brain originates; and that the occurrence of paralysis in connection with valvular lesions of the heart, may be thus accounted for in a certain proportion of

[1] *Vide* Brit. and For. Med.-Chir. Rev., July, 1857, p. 15. Virchow's earliest researches were in 1845. His later publications on this subject are contained in *Gesammelte Abhandlungen zur Wissenschaftlichen Medicin*, Frankfurt am Main, 1856, and *Handbuch der Speciallen Pathologie und Therapie*, vol. i., 1854. The reader will find a review of the first of these works in the No. of the *Brit. and For. Med.-Chir. Rev.* just referred to, and an able analytical review of the last in the *North American Med.-Chir. Rev.*, No. for July, 1858.

[2] "On some of the Principal Effects resulting from the Detachment of Fibrinous Deposits from the Interior of the Heart and their mixture with the Circulating Blood," by William Senhouse Kirkes, M. D., Trans. Med.-Chir. Society of London, 1852.

cases. Or, the embolus may follow the current downwards through the descending aorta, and become lodged in some one of the secondary branches. It seems to be sufficiently established that obstruction of the renal, splenic, iliac, femoral, and other arteries may be thus produced. Numbness, impaired muscular power of the lower extremities, loss of pulsation in the arteries accessible to the touch, and even gangrene, have been observed as probable results of obstruction of the iliac and femoral arteries.[1]

The production of arterial obstruction by detached deposits is rendered highly probable by the facts already stated, viz., the frequency with which loosely attached masses of variable size are observed on the valves in post-mortem examinations, and the occurrence of paralysis and other effects, in cases of valvular disease, which may fairly be attributed to this cause. But the proof rests mainly on the identity of the emboli or plugs found in the arteries with the deposits existing at the same time on the valves. To this point the attention of pathologists has been directed, and in numerous cases which have been reported this identity appears to have been sufficiently established. The fact of obstruction being ascertained, together with the existence of local changes (in the brain especially), which are apparently due to this obstruction, the obstructing mass has been found not only to resemble in its gross appearances the vegetations or warty excrescences coexisting within the heart, but to possess the same composition and formation as determined by microscopical examination. The plugs are sometimes calcareous, coexisting deposits within the heart having undergone a similar transformation.

Other effects attributed to the removal of deposits on the valves or orifices, and their transportation with the current of blood into the arteries, may be here alluded to. Instead of being detached in masses of greater or less size, they may be disintegrated and carried away in small particles. It is easy to conceive of a considerable quantity of the debris of fibrinous and calcareous deposits in this way accumulating within the vessels. This does not occa-

[1] Since this chapter was written, I have met with an instance of the formation of large masses of calcareous matter within the right ventricle, and the impaction of a mass as large as a pullet's egg in the left pulmonary artery. This division of the pulmonary artery was completely obstructed by the calcareous mass which had evidently been detached from within the ventricle, the fractured surface being apparent. Pulmonary tuberculosis coexisted in this case, the tuberculous deposit being most abundant in the right side.

sion palpable obstruction of arterial trunks like the plugs or emboli, but accumulating in the minute or capillary vessels in certain organs, local congestions and impaired nutrition may arise therefrom. Other and more serious consequences are imputed to the admixture of these particles with the blood. It is supposed that phenomena indicative of a morbid poison in the blood may be thereby induced. Cases in which typhoid symptoms, petechial eruptions, etc., occur in the course of heart affections are thus explained. This pathological view is, and must of necessity continue to be, hypothetical, since it is difficult, if not impossible, to demonstrate the presence of these particles, and their derivation from the heart. The doctrine has reference more to the recent products of inflammation deposited on the valves than to the deposits of long standing which characterize chronic valvular lesions. These deposits, from their consistency, are more likely to be detached in masses than to be disintegrated and carried away in fine particles, while soft lymph or fibrin is readily removed in the latter mode. Moreover, clinical observation shows that symptoms denoting blood poisoning very rarely occur during the progress of chronic valvular lesions.

Symptoms and Pathological Effects referable to the Respiratory System.

The phenomena referable to the lungs, in connection with valvular lesions, irrespective of associated or intercurrent pulmonary affections, depend, for the most part, on vascular engorgement of these organs. Congestion of the lungs is an immediate result of an impediment to the free admission of blood into the left auricle from the pulmonary veins. An impediment exists whenever the left auricle is over-distended with blood; and over-distension of this auricle occurs as a consequence of any interruption of the blood-currents through the orifices of the left side of the heart. These effects are involved more directly and in a greater degree in mitral than in aortic lesions. Obstructive lesions at the mitral orifice especially give rise to pulmonary congestion. The phenomena dependent thereon are most marked, other things being equal, in cases characterized by extreme contraction of this orifice. Mitral regurgitation leads to pulmonary congestion and its dependent phenomena, but not so readily nor to the same extent as in

cases of obstruction. The effects of regurgitation, however, are often added to those of contraction. Aortic lesions, obstructive and regurgitant, also, sooner or later, are followed by over-distension and dilatation of the left auricle and consequent congestion of the lungs. Dilatation of the left ventricle, however, precedes these effects, and the latter are produced more tardily than when the lesions are situated at the mitral orifice. In the rare instances of lesions occasioning obstruction or regurgitation at the tricuspid and pulmonic orifices, the over-distension of the right auricle which ensues constitutes an impediment to the circulation which affects the systemic vessels, not extending to the pulmonary vascular system unless the left ventricle becomes dilated. Engorgement of the lungs, therefore, is a special pathological effect of lesions affecting the orifices at the left side of the heart, as congestion of the systemic veins is, in like manner, a special pathological effect of an impediment existing in the right side. This statement applies to valvular lesions. It does not apply to another morbid condition which may contribute to pulmonary congestion, viz., dilatation or weakness of the right ventricle. The blood accumulates in the vessels of the lungs in consequence of not being propelled by this ventricle with sufficient power. Venous congestion is due to deficiency in the *vis à tergo*. Dilatation of the right ventricle, which is an effect of the engorgement of the lungs incident to mitral or aortic lesions, thus tends to augment the difficulty of the circulation through the pulmonary circuit. An enfeebled condition of the right ventricle is, in itself, adequate to produce pulmonary congestion, as is seen in some cases of fatty degeneration affecting this ventricle, but not to the extent of valvular lesions involving interruption of the blood-currents through the orifices in the left side of the heart. These remarks are alike applicable, *mutatis mutandis*, to dilatation or weakness of the left ventricle in connection with congestion of the systemic veins.

The engorgement of the lungs arising from valvular lesions gives rise to important pulmonary symptoms without any other superinduced affection of these organs. The most prominent of these symptoms are, dyspnœa, cough, muco-serous expectoration, and hæmoptysis. Certain pulmonary affections appear in some instances to be dependent directly and exclusively on over-distension of the vessels, viz., extravasation of blood, or apoplexy of the lungs, and œdema. Other affections are incidental to valvular lesions, the state of congestion predisposing to them, or favoring their devel-

opment. The existence of valvular lesions, thus, involves a liability to bronchitis, pneumonitis, pleurisy and emphysema.

Dyspnœa is a symptom more or less prominent in the great majority of cases. If there be no superinduced or incidental affection of the lungs, the difficulty of breathing is proportionate to the amount of pulmonary congestion. It is a criterion of the extent to which the changes effected by respiration are compromised in consequence of the retarded flow of blood through the capillary vessels. It occurs earlier and is more marked in cases of mitral than aortic lesions, because the former tend more directly and in a greater degree to engorgement of the pulmonary vessels. In most cases of either mitral obstruction or regurgitation, dyspnœa is the first symptom which occasions inconvenience. The patient often complains of this symptom alone, or chiefly, for a considerable period. In cases of aortic lesions it occurs later and is oftener preceded by palpitation or other symptoms referred by the patient to the heart. It is not uncommon to meet with examples of great contraction and insufficiency at the aortic orifice, accompanied by considerable enlargement of the left ventricle, when there had been little or no embarrassment of respiration. Instances are much less frequent of a similar amount of obstruction or regurgitation at the mitral orifice, which had not given rise to dyspnœa. When dependent on aortic lesions, this symptom is evidence of enlargement of the heart, since pulmonary engorgement does not occur until the left ventricle becomes dilated. When dependent on mitral lesions, dyspnœa may be experienced prior to much enlargement, the right ventricle becoming dilated or hypertrophied as an immediate result of the retarded circulation through the lungs. To the condition which the symptom represents, viz., pulmonary congestion, enlargement of the heart is thus antecedent in cases of aortic, and consecutive in cases of mitral lesions.

The intensity of dyspnœa varies greatly in the different cases of valvular affections in which this symptom is present, and in the same case at different periods. It consists, at first, of a slight deficiency of breath on exertion. This progressively increases until active exercise becomes insupportable. If the patient pursue an occupation which requires strong muscular movements, he finds it difficult, after a time, to continue them, and is at length compelled to give up labor. Cases frequently at this juncture first come under medical observation. The breathing may be sufficiently easy so long as quietude of the body is maintained, when the dyspnœa is

marked on taking moderate exercise, even walking across the room. Habitual dyspnœa, in some instances, does not occur, or it takes place only during the latter part of life. Other cases are characterized by paroxysms of difficult breathing when not provoked by exercise, and more or less difficulty may be apparent constantly. There is a notable difference in different cases as regards the consciousness of dyspnœa and the amount of suffering occasioned by the same apparent difficulty. The breathing is sometimes evidently labored when the patient makes no complaint, and says he experiences no inconvenience. This is probably owing, in part, to the symptom having been so gradually and imperceptibly developed that the mind becomes accustomed to it, and it is scarcely noticed so long as the habitual amount of difficulty only exists. The distress is not commensurate with the manifestations of difficulty, in other instances, because the perceptions are blunted by the circulation of imperfectly oxygenated blood. In these instances more or less lividity of the prolabia and surface of the body is apparent. The suffering, however, is often great. The want of more breath is painfully felt, amounting sometimes to a sense of suffocation. The patient cannot lie down, but is obliged to keep the sitting posture, often bending forward and supporting himself by the hands locked below the knees, or resting upon some solid body. The accessory muscles are brought into play, to produce the greatest possible expansion of the chest. The countenance expresses great anxiety, and frequently the lips and face are livid. Dyspnœa having this intensity is distinguished as *orthopnœa*. Occurring in paroxysms, it constitutes the *cardiac asthma* of writers.

These diversities as regards dyspnœa show that this symptom is affected by a variety of circumstances. As an objective symptom, its intensity corresponds to the amount of pulmonary congestion, provided no other affection of the lungs be present. Subjectively, its intensity depends, in a great measure, on the rapidity or slowness with which the pulmonary congestion has ensued: that is, the suffering incident to dyspnœa, when it is rapidly developed, is far greater than when it has been gradually induced. The striking difference among different persons in susceptibility to painful impressions serves also to explain the greater tolerance by some patients than by others of apparently an equal amount of difficulty of breathing. Affections of the pulmonary organs, superadded to congestion, contribute to increase the amount of dyspnœa. Emphysema, in connection with valvular lesions, in proportion to its extent, adds

intensity to this symptom. This combination is not infrequent, and the cardiac affection is liable to be overlooked unless due attention be directed to an examination of the heart. Pleuritic effusion increases the dyspnœa by diminishing the volume of lung and limiting the range of thoracic expansion. Bronchitis produces the same effect, the supply of air to the cells being diminished by swelling of the bronchial mucous membrane and the accumulation of mucus within the tubes. Œdema compromises the breathing capacity of the lungs in proportion as the air-cells become filled with effused liquid. The coexistence of these or other pulmonary affections of course invalidates, to a greater or less extent, the significance of dyspnœa as representing the amount of pulmonary congestion due to valvular lesions. It is only when pulmonary complications are excluded that this symptom is to be considered as a criterion of the impediment to the pulmonary circulation which these lesions occasion. Spasm of the muscular fibres of the bronchial mucous membrane—in other words, true asthma—may occur in connection with valvular lesions. This explains, in some instances, the occurrence of the paroxysms of dyspnœa or orthopnœa which constitute cardiac asthma. Various circumstances, however, which occasion, temporarily, a considerable increase of the pulmonary congestion, may give rise to these paroxysms. The disturbed action of the heart which generally exists during attacks of angina pectoris, renders dyspnœa, in some cases, an important element in that superadded affection.

Cough and expectoration are usually present when valvular lesions have induced considerable pulmonary engorgement. The congested state of the bronchial mucous membrane leads to an abnormal secretion of mucus, and transudation through the coats of the vessels into the tubes. The expectoration is muco-serous in its character. Varying in amount in different cases, it may be considerable without involving inflammation of the membrane. The affection, under these circumstances, is bronchorrhœa. But bronchitis is apt to become developed, giving rise to more cough, with an expectoration of modified mucus and muco-purulent sputa. The prominence of the cough and the characters which the expectoration presents, will serve to indicate, on the one hand, merely congestion and irritation of the bronchial membrane, or, on the other hand, a superinduced pulmonary affection. The existence and nature of the latter, however, are to be ascertained, not by the cough and expectoration alone, but by means of other associated

symptoms and by physical signs. The accumulation of liquid within the bronchial tubes often increases the dyspnœa, and, at an advanced period, may prove the immediate cause of death by asphyxia.

Hæmoptysis is a symptom which occurs in a pretty large proportion of cases of valvular lesions attended with a marked degree of engorgement of the lungs. It is due to distension of the vessels of the bronchial membrane; diminished cohesion from impaired nutrition eventuating in rupture at certain points. The amount of hemorrhage varies in different cases. It is rarely large, and often quite small. In some instances, frequent repetitions of the hæmoptysis take place. It is rare that the loss of blood is sufficient to constitute grounds for apprehension, although this symptom generally occasions alarm in the minds of patients and friends. The immediate effect is perhaps oftener salutary than otherwise, the pulmonary congestion being temporarily relieved by the direct depletion from the engorged vessels. Of the different lesions, mitral contraction is most likely to give rise to bronchial hemorrhage. It occurs, however, by no means exclusively in connection with this form, but is observed in cases of mitral regurgitation, and also in connection with lesions at the aortic orifice. It was formerly attributed incorrectly to hypertrophy of the right ventricle, the augmented power of the contraction of this ventricle being supposed to impel the current of blood into the pulmonary vessels with a force sufficient to produce rupture. It is not probable that this alone is ever adequate to give rise to hæmoptysis, but it is reasonable to suppose that it may exert some agency in conjunction with the valvular lesions which occasion obstruction at the left side of the heart. Dilatation of the right ventricle, however, by retarding the circulation through the lungs, probably co-operates with the valvular lesions in the production of this symptom, more than hypertrophy of this ventricle.

Hemorrhagic extravasation, pneumorrhagia, or pulmonary apoplexy, involves the same pathological explanation as hæmoptysis, but occurs much more unfrequently than the latter. It is an occasional effect of engorgement. In most of the cases in which it occurs, there exists mitral contraction. This pulmonary affection is very rarely observed except in connection with valvular lesions which give rise to obstruction at the left side of the heart. As regards the agency of hypertrophy of the right ventricle in its production, the remarks made with reference to hæmoptysis are alike

applicable. Hæmoptysis and hemorrhagic extravasation occasionally co-exist, but the latter, as well as the former, occurs without the other. In proportion to the extent of solidification of lung by the extravasated blood, will the respiratory function be compromised, and dyspnœa increased. The symptoms and signs pertaining to this affection will, of course, be superadded to those which belong to pulmonary congestion. For the diagnostic points, which are not highly distinctive, the reader is referred to works treating of diseases of the respiratory system.

Pulmonary œdema is another pathological effect attributable directly to over-distension of the vessels of the lungs. This event takes place much more frequently than extravasation of blood. The liability to its occurrence, other things being equal, is proportionate to the amount of obstruction at the left side of the heart; but it is more likely to occur when dilatation, or weakness of the right ventricle is superadded. A condition of the blood disposing to transudation favors its occurrence. When such a condition exists, œdema of the lungs occurs in connection with effusion in other situations, or general dropsy. Occurring alone, or irrespective of dropsical effusion elsewhere, it belongs among the events incident to an advanced stage of valvular lesions. It adds to the dyspnœa in proportion to the amount of pulmonary parenchyma involved, and also increases the cough and expectoration. In a certain proportion of cases it proves the immediate cause of death by asphyxia. Its occurrence is denoted by physical signs (dulness on percussion and the subcrepitant or a fine mucous rale), which generally render practicable the diagnosis.

The symptoms and pathological effects which have been noticed, it will be borne in mind, are produced by valvular lesions through the intervention of the pulmonary engorgement incident to these lesions. They are immediate effects of this engorgement. Valvular lesions, therefore, may exist without giving rise to these effects, so long as the lesions do not interfere materially with the pulmonary circulation. It does not follow from the fact that there is abundant evidence of the existence of valvular lesions, that these symptoms and effects will speedily occur, for lesions may exist for an indefinite period without occasioning a marked degree of congestion of the lungs. Moreover, the pulmonary circulation bears with impunity a certain amount of obstruction. As a rule, whenever events of importance referable to the respiratory system become developed, valvular lesions have existed for a considerable length of time, and

have led to more or less enlargement of the heart. The heart, when enlarged, with or without lesions of the valves, encroaches on space which otherwise would have been occupied by the lungs. In this way the respiratory function is to some extent compromised. I have met with instances of collapse of the lower lobe of the left lung, apparently due to the pressure of an enlarged heart.

Certain pulmonary affections not due directly or exclusively to the congestion proceeding from valvular lesions, are more apt to occur under these circumstances, than if the latter did not exist. The lesions thus indirectly predispose to the development of these affections. Emphysema of the lungs is one of these affections. This is certainly observed among a given number of persons affected with valvular lesions, in a larger ratio than among the same number of persons free from these lesions. Without entering into a discussion of the mechanism by which pulmonary emphysema is produced, which would be here out of place, I shall simply remark that the histories of cases in which this affection is developed during the progress of valvular lesions show that it is preceded and accompanied by bronchitis, to which it probably sustains the same relation as when it is developed irrespective of cardiac disease. Occurring as a complication of valvular lesions, it adds notably to the dyspnœa, overshadowing in some instances the cardiac symptoms. Moreover, increasing the obstruction to the pulmonary circulation, it co-operates with the impediment due to the valvular lesions in leading to enlargement of the right ventricle. It is not easy in individual cases always to determine the amount of dyspnœa and other symptoms attributable, on the one hand, to the emphysema, and, on the other hand, to the congestion incident to valvular lesions. This problem can only be solved approximately by endeavoring to estimate the extent to which the lungs are emphysematous, by means of diagnostic signs which it belongs to works treating of diseases of the respiratory system to consider, and also ascertaining the amount of cardiac enlargement which exists.

The congested state of the bronchial mucous membrane renders it prone to inflammation. Bronchitis is a frequent complication of valvular lesions after they have induced pulmonary engorgement. This complication occurring in persons free from antecedent disease of the lungs or heart, and limited, as is usually the case, to the larger tubes, very rarely gives rise to dyspnœa. But developed when the lungs are already congested in connection with cardiac lesions, dyspnœa becomes more or less prominent. The existence

of bronchitis, disconnected from other pulmonary affections, is determined by its positive symptoms and signs, and by the absence of the diagnostic phenomena which characterize other affections. The coexistence of bronchitis not only adds to the distress incident to valvular lesions which interfere with the pulmonary circulation, but, if severe or extensive, often places the patient in immediate danger, the accumulation of the products within the bronchial tubes, together with the diminished calibre of the tubes from swelling of the membrane, inducing suffocation. In some persons the bronchial inflammation leads to spasm of the muscular fibres of the membrane, giving rise to attacks of true asthma. As already stated, it is probably by the intervention of bronchitis that valvular lesions predispose to emphysema.

Persons affected with valvular lesions which have led to pulmonary congestion, are more prone than others to pneumonitis. The affection occurring in this connection is attended with much greater embarrassment of respiration, and disturbance of the circulation, than when it occurs as a primary disease; and it is more likely to prove fatal. Death sometimes occurs during the first stage of the disease, which is very rarely the case when pneumonitis is primitive. The characteristic symptoms and signs of the affection render the fact of its coexistence, in individual cases, easily determinable.

Dropsical effusion into the pleural sacs rarely occurs to much extent independently of general dropsy. When it does take place, the compression of the lungs by the effused liquid abridges their functional capacity, aggravates the dyspnœa, and hastens a fatal issue. The effusion, when purely dropsical, exists in both sides of the chest; the quantity in one side, however, often exceeding considerably that in the other side. But, in a certain proportion of cases, pleuritic inflammation becomes developed in one side, giving rise to an abundant effusion of liquid, nearly or quite filling the affected side. In several instances which have fallen under my observation, pleurisy has occurred when the cardiac affection had not previously occasioned sufficient inconvenience to prevent the patients from continuing laborious occupations. The inflammation is subacute. The affection is developed, as cases of chronic pleurisy frequently are, with little or no pain, the chief subject of complaint being dyspnœa. So slight are the symptoms referable to the lungs, exclusive of dyspnœa, that if the attention of the physician be directed to the heart, there is a liability of overlooking

the pleuritic effusion, unless pains are taken to explore the chest for the signs of this affection. On examination after death the liquid effused is not transparent as in cases of hydrothorax, but not notably turbid, and the solidified fibrin, or lymph, is not abundant. The inflammatory action has a low grade of intensity. This is true of the instances that have come under my observation. The effused liquid is less likely to be removed by absorption than in cases in which chronic pleurisy is not connected with valvular lesions. A fatal termination is hastened by this complication, and in some instances death takes place very soon after its occurrence. The physical signs of an abundant pleuritic effusion are so simple and well marked, that if overlooked by those who avail themselves of physical exploration of the chest, it must be from inattention. Chronic pleurisy is developed in some cases in which valvular lesions are associated with albuminuria and Bright's disease, but it occurs when the kidneys are free from disease.

Symptoms and Pathological Effects referable to the Nervous System.

The majority of cases of valvular lesions end without having given rise to prominent symptoms or important pathological effects referable to the nervous system. This statement is at variance with the notions generally entertained and inculcated by some writers. It is a common impression that various symptoms denoting cerebral disorder, such as cephalalgia, vertigo, tinnitus aurium, muscæ volitantes, etc. etc., are usually observed, sooner or later, during the progress of cardiac disease. These symptoms are often observed in persons not affected with disease of the heart, and, hence, would possess small diagnostic significance were they more frequently present; but, the truth is, they occur in only a small proportion of cases, at least in a marked degree. Apoplexy and paralysis are events much less common than is generally supposed. It is then hardly necessary to consider at much length the mooted question, whether cerebral phenomena and complications proceed from the abnormal power of an hypertrophied left ventricle, or from the obstruction occasioned by over-distension of the right auricle. It is conceivable that either and both may conduce, in certain instances, to congestion, extravasation of blood, and serous transudation; but clinical facts show that, separately or combined,

they very rarely produce disorder of the cerebral circulation sufficient to occasion great inconvenience to the patient or lead to serious results. Of seventy-two fatal cases of lesions affecting the mitral and aortic orifices, one or both, which I have analyzed with reference to this point, the histories of fifty-five present no symptoms or events of importance pertaining to the brain.

Valvular lesions, accompanied by enlargement of the heart, have been supposed to involve a strong liability to apoplexy. This opinion was held by Hope. Apoplexy occurs in a small proportion of cases of valvular disease. Of the seventy-two cases analyzed it took place in seven. But even in the few cases in which apoplexy and valvular lesions are associated, circumstances go to show that there often exists only a remote and contingent pathological connection between them. Of the seven cases just mentioned, the age of the patients in all, save one, was over forty years. In one, it was eighty, in one, sixty-six, in one, fifty-five, and in one, fifty-two. The ages were within the period when apoplexy, irrespective of heart disease, is most apt to occur. In some of the cases, the cerebral arteries were found to have become calcified. In one of the cases, the patient being forty-one years of age, the valvular affection was trivial, accompanied by slight enlargement. It is not unfrequently the case when, from the coincidence of apoplexy and some morbid appearances of the valves, the former is supposed to be dependent on the latter, that the valvular lesions are not sufficient to have occasioned much disturbance of the circulation. On the other hand, how rarely does apoplexy occur when there exists over-distension and dilatation of the right auricle, together with considerable hypertrophy of the left ventricle! In view of these facts, it is reasonable to conclude that apoplexy is very rarely due, directly or exclusively, to the condition of the heart, but that the changes which the cerebral vessels undergo, or other circumstances, generally play an important part in its production. This is not to deny more or less agency to the heart in certain cases. And of the two conditions which tend directly to affect the circulation in the brain, viz: obstruction at the right side of the heart, and hypertrophy of the left ventricle, the former must be considered as most likely to lead to serious results. Hypertrophy of the left ventricle is generally associated either with obstruction or regurgitation at the aortic or mitral orifice. An effect of each of these different lesions is either to diminish the quantity of blood sent to the brain, or to break the force of the ventricle upon the arterial current.

The latter obtains when the aortic orifice is contracted, and the former in cases of aortic regurgitation and of mitral lesions, whether obstructive or regurgitant. The opinion held by Hope and others that apoplexy sustains a direct pathological connection with hypertrophy of the left ventricle, is disproved on rational grounds as well as by clinical evidence.

Apoplexy occurring in connection with cardiac lesions generally depends on extravasation of blood. Under these circumstances, paralysis, of course, ensues. If the apoplectic attack do not prove suddenly fatal, the patient is found to be hemiplegic. Paralysis sometimes occurs without being preceded by apoplexy. Either or both may proceed from a cause emanating from the heart, independently of either an impediment at the right auricle or hypertrophy of the left ventricle. Reference is now made to an event which has been already noticed under the head of pathological effects referable to the circulation, viz., detached fibrinous deposits or emboli. It appears to be sufficiently established that these sometimes become fixed in the arterial trunks of the brain, and give rise to apoplectic seizures with or without paralysis, or to the latter without the former. In this way, valvular lesions may sustain towards these cerebral affections a direct causative relation. This explanation of apoplexy and paralysis associated with valvular lesions is rendered probable in cases in which, from the age of the patient, fatty or calcareous degeneration of the cerebral arteries is not likely to have occurred, and when there does not exist a notable degree of obstruction at the right side of the heart. After death, this is to be suspected when the aortic or mitral valves are found to present vegetations or excrescences, some of which are loosely attached. The proof consists in finding deposits or emboli impacted in one or more of the arterial trunks of the brain, which are found, on microscopical examination, to have the same composition and interior arrangement as coexisting deposits on the valves of the left side of the heart. Complete recovery from paralysis is a ground for suspecting that it originated in arterial obstruction rather than in extravasation, the restoration of power over the paralyzed muscles, when the latter occurs, being rarely perfect.

Arterial obstruction is supposed to give rise to apoplectic phenomena and paralysis by lessening the supply of blood to certain portions of the cerebral substance. The pathological condition induced, therefore, is the opposite of that incident to an impediment to the circulation at the right side of the heart. In the one case

a part of the brain suffers from anæmia, in the other case the whole brain is congested. Softening of the cerebral substance has been observed in connection with the interruption of the circulation by fibrinous plugs or emboli, attributable to impaired nutrition from the defective supply of blood. Dr. Law, of Dublin, attaches considerable importance to the defective supply of blood to the whole brain as a result of either mitral or aortic obstruction. In these lesions more especially, but to a considerable extent also in those attended by regurgitation, the stream of blood propelled into the aorta and cerebral arteries is obviously lessened. The brain receives with each contraction of the left ventricle a quantity of blood less than when the valves and orifices are free from disease. Dr. Law attributes cerebral softening in some instances to this cause.[1] The importance attached by this writer, however, to an anæmic state of the brain, as thus induced, is not sustained by clinical observation, since it is only in a small proportion of the cases attended with more or less obstruction or regurgitation, or both, at either the aortic or mitral orifice, that cerebral symptoms denoting any important pathological condition are manifested. Cases of extreme aortic and mitral contraction proceed to a fatal termination, the histories not containing aught which indicates that the brain has suffered from a deficient supply of blood.

Attacks of pseudo-apoplexy, that is, of coma, more or less complete, continuing for a certain period and passing off without paralysis, have been observed in cases of valvular lesions. They have been already described in treating of fatty degeneration of the heart. They may depend on the latter change coexisting with valvular lesions; but, as remarked in connection with the subject of fatty degeneration, the pathological relation between these attacks and the existence of any organic disease of the heart cannot be considered as established.

Aside from apoplexy and paralysis, the various symptoms already mentioned, viz., pain, vertigo, tinnitus, etc., are occasionally associated with valvular lesions. Apoplexy and paralysis depending either on an extravasation which involves a morbid condition of the cerebral vessels, or on arterial obstruction from emboli, are usually not preceded by premonitions referable to the brain. Clinical observation shows that a liability to these affections is not to be predicated on the symptoms just referred to. This is a practical

[1] Dublin Journal of Medicine, May, 1840.

point to be borne in mind in order that gratuitous apprehensions need not be entertained on the part of physician or patient, and measures employed, with a view of warding off an attack of apoplexy or paralysis, which, being uncalled for, will be likely to be not only unnecessary, but injudicious. In the cases in which there must be more or less cerebral congestion, the superficial veins of the neck being swelled or pulsating, marked cerebral symptoms are not uniformly present. Headache, dulness of the intellect, listlessness, drowsiness, etc., are symptoms which, in a certain proportion of cases of this description, are more or less marked, and are probably due to abnormal fulness of the cerebral veins. These symptoms of cerebral oppression are sometimes marked in cases in which, either from obstruction at the right side of the heart or imperfect oxygenation of the blood, the prolabia and surface of the body present a livid appearance.

The sleep of patients affected with cardiac disease is frequently imperfect. They complain sometimes of frightful dreams. This is generally associated with dyspnœa, and appears to be owing to disturbed respiration rather than to disordered cerebral circulation. Moaning in sleep is a symptom observed in some cases, when the patient is not wakeful nor conscious of any morbid sensations.

A symptom which may be included among the events referable either to the nervous or respiratory system, is noticed in some cases, viz: a choking sensation analogous to that experienced in painful emotions when an effort is made to refrain from weeping. This is not of frequent occurrence, but it has been prominent in several instances among the cases that have come under my observation. It is associated with more or less dyspnœa.

The mental condition of patients affected with organic disease of heart, may be noticed in this connection. The contrast presented in this respect, with patients affected with merely functional disorder, has been already referred to. Persons with organic disease which has given rise to grave symptoms, such as palpitation, dyspnœa, dropsy, etc., are generally free from excitement and apprehension. They often seem to be remarkably indifferent or apathetic. They are not agitated when made acquainted with the fact that they have organic disease of the heart. They are sometimes incredulous as to the seat of the disease, and are disposed to attribute their ailments to the liver, lungs, or stomach. The mental condition, in short, is quite the reverse of that usually associated with affections purely functional. It is not unlike that which exists in connection

with pulmonary tuberculosis. A comparison of the characters pertaining to the feelings, which belong to the history of organic lesions of the heart, with those observed in some other diseases, affords a striking illustration of the great difference in the effects produced on the mind by different morbid conditions irrespective of cerebral diseases.

Some degree of mental aberration is occasionally observed toward the close of life in cases of valvular lesions, but delirium cannot be reckoned among the events belonging to their natural history.

Symptoms and Pathological Effects referable to the Digestive System and Nutrition.

The phenomena manifested in connection with the digestive apparatus in cases of valvular lesions, proceed from congestion of the systemic venous system. Assuming the lesions to be either mitral or aortic, or both, congestion of this order of vessels depends on the effects of these lesions on the right side of the heart. It may be stated that, as a rule, the systemic congestion is not sufficient to give rise to important symptoms or pathological effects until dilatation of the right ventricle has taken place, involving overdistension of the right auricle, and, in certain instances, tricuspid regurgitation. The impediment to the free admission of blood from the venæ cavæ into the right auricle, occasions cerebral congestion, as has just been seen. The congestion throughout the body thus induced, as has also been seen, gives rise to venous turgescence and general dropsy. The abdominal viscera indirectly participate in the effects of this impediment at the right ventricle, owing to their vascular relations to the venæ cavæ being through the intervention of the portal system. In view of the anatomical peculiarities of the latter, it is obvious that, of the organs comprising the abdominal viscera, the liver is first affected by an obstruction at the right side of the heart. The radicles of the hepatic veins (the intra-lobular veins) are the first of the different orders of vessels contained in this viscus, to show engorgement. The terminal branches of the portal vein (the inter-lobular veins) are next affected. The appearances after death indicate whether either or both of these sets of vessels are unduly congested. The pressure of the portal branches, or inter-lobular veins, on the

biliary tubes may occasion an undue accumulation of bile in the latter. Sections of the organ then present that peculiar aspect commonly known as the "nutmeg liver." Extending beyond the liver to the portal vein and its radicles, the congestion affects finally the stomach and intestines, the spleen and the pancreas. Congestion of these organs is a secondary effect due directly to the mechanical obstacle to the passage of blood through the liver. The successive steps, thus, in the series of congestive effects dependent on valvular lesions are: Obstruction or regurgitation at either the mitral or aortic orifice, or at both situations; dilatation of the right ventricle following engorgement of the pulmonary vessels; overdistension of the right auricle, with or without tricuspid regurgitation, involving an impediment to the free transmission of blood from the venæ cavæ; congestion of the hepatic vein, and its radicles, the intra-lobular veins; congestion of the terminal branches of the portal vein, or the inter-lobular veins; congestion of the vena portæ and its radicles in the abdominal viscera which furnish the blood for the portal circulation.

Clinical observation shows, as might rationally be anticipated, that the phenomena due to engorgement of the abdominal viscera, are developed, in the order of time, consecutively to the general effects of congestion of the systemic veins. It is rarely the case that the former occur to much extent until the obstruction at the right side of the heart is sufficient to give rise to more or less general dropsy. As a general remark, symptoms and pathological effects referable to the digestive system do not hold a prominent place among the events which belong to the natural history of valvular lesions. This statement is made after analyzing the histories of one hundred cases, extending in seventy to the period of death. In a large majority of these histories, nothing of importance was noted with reference to the digestive system.

Enlargement of the liver is an occasional effect incident to valvular lesions, as well as to enlargement of the heart, uncomplicated with the latter. This has been already noticed in connection with the subject of enlargement of the heart. It is more correct to say that this is an effect of enlargement affecting the right side of the heart, either with or without the coexistence of valvular lesions, the latter inducing the effect through the intervention of the cardiac enlargement, as has just been stated. The augmented size of the liver is in some instances remarkable, and its variations in size at

different periods is not less striking. The enlargement is due simply to the excessive accumulation of blood in the vessels of the organ. Jaundice is an occasional symptom. It is met with, however, in a very small proportion of cases.

Cirrhosis, contrary to a common impression, is not a frequent complication of valvular affections of the heart. The congested state of the liver incident to these affections does not seem to tend to its production. The concurrence of these affections and this structural change of the liver is so infrequent, as hardly to afford ground for the opinion that there exists between the two any pathological connection. When associated, it is probably simply a coincidence. M. Becquerel reported the existence of cardiac disease in twenty-one of forty-two cases of cirrhosis which he analyzed, the former being deemed to have occurred prior to the latter.[1] But in more than one-half of the twenty-one cases, he regarded the cirrhosis as in the first degree giving rise to no symptoms of importance. These statistics, as remarked by Dr. Budd, are to be accounted for on the supposition that the abnormal appearances due to congestion of the different sets of vessels were confounded with the commencement of cirrhosis. The symptom denoting coexisting cirrhosis, is ascites in a degree disproportionate to the general dropsy. So far, however, from there being often a preponderance of ascites, it is generally less than the relative amount of dropsical effusion into the pleural cavities in cases of valvular lesions of the heart.

The various phenomena included in the term indigestion, which are common to a great number of affections, may be absent or present, and more or less prominent in cases of valvular lesions. But in a large proportion of cases they do not occur in a marked degree, at least during the greater portion of the time occupied by the progress of the lesions before a fatal issue takes place. Patients who suffer from the distressing effects of obstructive or regurgitant lesions, together with enlargement of the heart, often preserve their appetite, and the ingestion of food occasions no inconvenience. This, in fact, is usually the case, so that disordered digestion cannot be considered to characterize organic affections of the heart. The disorders which are observed in an advanced stage, after general dropsy has taken place, are probably due, in part at least, to con-

[1] Archives Générales de Médecine, 1840. Budd on Diseases of the Liver, second Am. ed., p. 148.

gestion of the gastric mucous membrane. Hæmatemesis is one of the rare effects, occurring sometimes when cirrhosis of the liver does not coexist.

Intestinal flux, or enterorrhœa, is another infrequent symptom, the serous transudation taking a direction through the mucous tissue, instead of, or in addition to the more common direction into the peritoneal cavity.

Hemorrhage from the bowels is to be ranked in the same category. The same is to be said of hæmorrhoids. In this connection may be mentioned epistaxis, which occurs more frequently, as a result of obstruction at the right side of the heart, than hemorrhage in any other situation. It is supposed that the escape of blood from the nostrils may in some instances prevent extravasation into the brain, or other serious effects of cerebral congestion, by relieving the vessels, in a measure, of their over-accumulation.

Enlargement of the spleen, due exclusively to the congestion of the portal system dependent on cardiac obstruction, must be exceedingly rare. It is, however, to be reckoned among the effects which are occasionally observed.

The functions of nutrition, applying this term to the processes of growth and repair of the tissues, are much less affected than, on rational grounds, would be anticipated, even when the lesions of the valves have led to enlargement of the heart and much disturbance of the circulation. Patients suffering from the distressing effects of cardiac disease, viz., dyspnœa, palpitation, œdema, etc., often do not emaciate. When these effects occur in early life, the development of the body is sometimes not remarkably impaired. Even at an advanced stage, considerable embonpoint is frequently maintained. It is not uncommon to find the evidences of lesions, which must have existed for a long time, in persons whose general aspect denotes excellent health. So far from diminished nutrition being one of the pathological effects of valvular lesions, they are rather to be characterized by the absence of notable deterioration in this respect. In cases in which the origin of valvular lesions dates in early life, and enlargement of the heart takes place before puberty, the body may attain to a full development.

Symptoms and Pathological Effects referable to the Genito-urinary System.

The renal or emulgent veins terminating in the vena cava descendens, the kidneys must participate in the congestion of the systemic venous system arising from an impediment at the right side of the heart. These organs are affected more directly than the abdominal viscera which are tributary to the portal vein. So soon as valvular lesions have led to the anatomical conditions involving an obstruction extending to the venæ cavæ and their branches, renal engorgement necessarily ensues. Congestion of these organs is generally observed in examinations after death in cases of valvular disease accompanied by dilatation of the cavities of the right side of the heart. Venous congestion, under these circumstances, does not uniformly occasion a greater flow of urine than in health. Indeed, the quantity of urine is oftener diminished than increased, a fact going to show that diuresis depends on the amount of blood conveyed to the kidneys by the arteries, or on conditions pertaining to the blood itself, rather than on accumulation in the renal veins. The urine is frequently scanty, even when the venous obstruction is sufficient to give rise to general dropsy. The solid constituents are relatively augmented; in other words, the density of the urine is greater than in health. The lithatic deposits are often abundant. The presence of albumen is not unfrequently shown by the appropriate tests. If the kidneys have not undergone structural change, the quantity of albumen is usually slight. It may be found, on repeated examinations in the same case, sometimes present, and at other times absent. The quantity at different times may be found to fluctuate. The presence of this constituent in these cases may be fairly attributed to the mechanical pressure incident to venous congestion. It does not constitute evidence of structural change of kidney or Bright's disease when it is in small quantity, transient in duration, and notably fluctuating.

The degenerations of structure included under the name of Bright's disease are sometimes associated with valvular lesions of the heart. The frequency of this combination, however, is less than is generally supposed. Accepting, on the one hand, as evidence of coexisting Bright's disease an abundant quantity of albumen constantly present in the urine during life, or well-marked anatomical characters observed after death, and, on the other hand,

including cases only of cardiac disease which involve obstruction or regurgitation, or both, the two affections are rarely united. It may fairly be doubted whether they are associated sufficiently often to establish any direct pathological connection between them. Assuming the existence of such a connection, some have attributed the renal affection to the cardiac disease, and others have thought that the valvular lesions were due to the condition of the kidneys. They who accept the first of these suppositions refer the development of structural change in the kidneys to the congested state of these organs. But it is by no means settled that mere congestion is adequate to produce this result. The second supposition is more tenable. Pericarditis and other serous inflammations are not unfrequently developed in the course of Bright's disease; and it may be reasonably argued that endocarditis is occasionally incidental to the latter. Clinical observation has not, as yet, confirmed the correctness of this analogical argument.

The coexistence of structural degeneration of the kidney is shown, as already intimated, by the degree and constancy of the albuminuria, and by the different varieties of casts of the uriniferous tubes, distinctive of the different kinds of degeneration, which the sediment of the urine is found to contain when subjected to microscopical examination.[1] The tendency to general dropsy is augmented by this complication; renal and cardiac dropsy are, in fact, combined. It is needless to say that the danger is vastly increased by the addition of so serious an affection as structural degeneration of the kidneys, which exposes the patient to other accidents than those incident to the cardiac disease, and aggravates some of the most important of the pathological effects of the latter.

As regards the generative functions, the histories of valvular affections which I have collected furnish no facts of significance or importance. I have observed, in cases in which lesions had existed for a considerable period before puberty, that the genital organs, including, in females, the mammary gland, have attained to a full development.

[1] *Vide* Diseases of the Kidney, etc., by George Johnson, M. D., etc. Lond., 1852.

Symptoms and Pathological Effects referable to the Countenance and External Appearance of the Body.

The characters pertaining to the countenance have, for the most part, been already incidentally mentioned. Lividity of the prolabia, face, and, to some extent, apparent over the whole surface of the body, denotes either venous congestion or imperfect oxygenation of the blood. The latter is incident to the pathological effects taking place in the lungs; the former, to obstruction at the right side of the heart. But both conditions may be conjoined. Cyanosis dependent on congenital malformations will be considered hereafter. A dusky hue of the face, combined with œdema, is quite distinctive of cardiac, as contrasted with renal, dropsy. The experienced clinical observer is able to make this differential diagnosis with much precision at a glance. When the lividity is marked, and the œdema considerable, the face presents an appearance like that of a cadaver after strangulation. The expression is sometimes so much altered that the person is scarcely recognized by familiar friends. Urgent dyspnœa induces an expression of great anxiety, distress, and apprehension. The painful spectacle presented by a case of extreme suffering from so-called cardiac asthma is thus vividly portrayed by Dr. Hope: "Incapable of lying down, he is seen for weeks, and even for months together, either reclining in the semi-erect posture supported by pillows, or sitting with the trunk bent forward and the elbows or forearms resting on the drawn-up knees. The latter position he assumes when attacked by a paroxysm of dyspnœa; sometimes, however, extending the arms against the bed on either side, to afford a firmer fulcrum for the muscles of respiration. With eyes widely expanded and starting, eyebrows raised, nostrils dilated, a ghastly and haggard countenance, and the head thrown back at every inspiration, he casts around a hurried, distracted look of horror, of anguish, and of supplication: now imploring in plaintive moans, or quick, broken accents, and half-stifled voice, the assistance already often lavished in vain; now upbraiding the impotency of medicine; and now, in an agony of despair, drooping his head on his chest, and muttering a fervent invocation for death to put a period to his sufferings. For a few hours—perhaps only for a few moments—he tastes an interval of delicious respite, which cheers him with the hope that the worst is over, and that his recovery is at hand. Soon that hope vanishes.

From a slumber fraught with the horrors of a hideous dream he starts up with a wild exclamation that 'it is returning.' At length, after reiterated recurrences of the same attacks, the muscles of respiration, subdued by efforts which the instinct of self-preservation alone renders them capable of, participate in the general exhaustion, and refuse to perform their function. The patient gasps, sinks, and expires."[1] Happily, the fearful intensity of suffering depicted in the foregoing sketch characterizes a small proportion only of the cases of valvular disease which proceed to a fatal termination.

Some cases of valvular disease are characterized by pallor of the complexion. The coexistence of Bright's disease is likely to lead to this effect. But it is observed in some instances when the kidneys are not affected. It then depends on alterations of the blood proceeding from other causes. According to MM. Becquerel and Rodier, an anæmic condition is induced, in a certain proportion of cases, by cardiac disease uncomplicated with an affection of the kidneys.[2] Analysis of the blood shows a notable deficiency of albumen, together with a reduction in the relative proportion of blood-corpuscles and fibrin. This condition of the blood is important in connection with therapeutical measures. It will be aggravated by depletion, and to remove it by appropriate treatment, if practicable, should be an important object with the practitioner.

The accumulation of blood in the right chambers of the heart induces, in addition to abnormal fulness of the superficial veins, a congestive state of the capillary vessels, causing the surface of the body to present an appearance like that produced by exposure to cold. The redness disappears on pressure, and returns, more or less slowly, after the pressure is removed. The appearance is not unlike that observed in the typhus and typhoid fevers, although the *rationale* is by no means the same. Erythema affecting portions of the surface occurs in some cases, not associated with œdema. The lower extremities are most apt to be affected. I have met with an instance in which the extremities of the fingers and a portion of the palms presented permanently an erythematic redness. On the other hand, in a patient with considerable mitral regurgitation, the fingers at times are bloodless, being as pallid and cold as those of a corpse.

[1] On Diseases of the Heart, Am. ed., p. 382.

[2] Gazette Médicale de Paris (13 Avril, 1850). *Vide* Précis théorique et pratique, par C. Forget.

CHAPTER IV.

PHYSICAL SIGNS, DIAGNOSIS, AND TREATMENT OF VALVULAR LESIONS.

Endocardial or valvular murmurs—Distinction between endocardial and exocardial murmurs—Vascular murmurs—Distinction between inorganic and organic murmurs—Soft and rough murmurs—Musical murmurs—Enumeration of abnormal conditions giving rise to endocardial murmurs, organic and inorganic—Valvular lesions involving obstruction or regurgitation, or both, generally accompanied by a murmur—Circumstances enabling the auscultator to determine whether lesions involve obstruction, or regurgitation, or both—Mitral direct, or systolic murmur—Mitral regurgitant, or diastolic murmur—Aortic direct, or systolic murmur—Aortic regurgitant, or diastolic murmur—Localization of systolic murmurs—Localization of diastolic murmurs—Recapitulation of points involved in the localization of systolic and diastolic murmurs—Pathological import of organic endocardial murmurs—Inorganic murmurs—Abnormal modifications of the heart-sounds in cases of valvular lesions.—Purring tremor—Diagnostic characters of mitral, aortic, tricuspid, and pulmonic lesions—Treatment of valvular lesions.

The physical signs of lesions affecting the valves and orifices of the heart are to be considered preparatory to entering on the consideration of the diagnosis of these lesions. It is chiefly by means of physical signs that the existence and seat of valvular lesions are determined during life. The symptoms and pathological effects which were considered in the last chapter, afford important aid to the diagnostician, but, alone, they often fail in furnishing positive evidence that the valves or orifices are affected, and still less do they indicate the particular situation of existing lesions. To the study of the physical signs, in fact, in connection with researches on the mechanism of the heart's action, practical medicine is indebted for the great perfection to which the diagnosis of cardiac affections has attained within the past few years. Here, as in other instances, physical phenomena have a negative as well as positive application in diagnosis; that is to say, while they constitute evidence of the presence of certain lesions, their absence is, in general, proof that lesions do not exist.

The diagnostic signs of valvular lesions are, for the most part, obtained by auscultation. They may be arranged into two classes,

viz: *first*, certain new or adventitious sounds called *murmurs;* and, *second*, abnormal modifications of the natural cardiac sounds. The first class, or murmurs, from their practical importance, require to be considered at some length.

ENDOCARDIAL OR VALVULAR MURMURS.

All adventitious sounds dependent on the movements of the heart, either replacing or superadded to the normal heart-sounds, are distinguished as *cardiac murmurs*. Strictly speaking, these murmurs are heart-sounds, but, for the sake of distinction, the latter term is conventionally restricted to the normal sounds of the heart and their abnormal modifications. The French word *bruit* is often used by English and American writers. This term with French writers is synonymous with murmur. The latter term, first proposed by Dr. Forbes, of London, is sufficiently distinctive and convenient, so that it is quite needless, in this instance, to have recourse to a foreign tongue.

Cardiac murmurs originate either within the heart and blood-vessels, or on the peripheral surface of the organ. Dr. Latham has proposed to distinguish those produced within the heart as *endocardial*, and those produced upon the surface as *exocardial* murmurs. These names are sufficiently distinctive. It is, however, convenient to include among endocardial murmurs those produced in the aorta and pulmonary artery in close proximity to the heart. Murmurs produced within vessels more or less removed from the heart, may be called *vascular murmurs*. These may originate either within the arteries or veins. The endocardial and vascular murmurs require for their production the passage of currents of blood through the cavities of the heart, its orifices or the bloodvessels. These murmurs are generally called *bellows murmurs* (*bruit de soufflet*) from the resemblance of the sound to that produced by the expulsion of air from the nozzle of an ordinary bellows. This resemblance is often striking, and holds good in most instances; but some intra-cardiac and vascular murmurs are very inappropriately called bellows-murmurs, resembling other sounds more than that implied by this name. It is, however, to be borne in mind that all adventitious sounds produced within the heart and vascular system, are conven-

tionally known as bellows murmurs. This name was applied to them by the illustrious discoverer of auscultation, Laennec, by whom they were first described.

Exocardial murmurs are occasioned by the rubbing together of the visceral and parietal surfaces of the pericardium, and sometimes by the heart impinging against the neighboring parts exterior to the pericardium. These murmurs are usually called, from the manner of their production, *attrition* or *friction murmurs.* These will be considered hereafter in connection with the diseases affecting the pericardium.

In treating of endocardial murmurs, the practical points to be considered relate to the different characters which they present, the morbid conditions which they denote, their significance and value as signs of disease, and their application to the diagnosis of valvular affections. These murmurs may be produced within the cavities of the heart, at the auriculo-ventricular or the ventriculo-arterial orifices, and within the aorta or pulmonary artery near the junction of these vessels with the ventricles. Strictly speaking, murmurs produced in the last-named situations are not endocardial, but it is most convenient to include them in the same class. It is practicable often, if not generally, to determine by means of physical exploration, in which one of the cavities, orifices, or vessels mentioned, originate the murmurs heard in individual cases. The importance of this localization, as pointing to the seat of the lesions which occasion the murmurs, is obvious. The special objects to which the study of this class of murmurs has been subservient, may be stated as follows: To determine the existence or non-existence of valvular disease; to determine the particular situation of structural lesions; to determine the character of lesions and certain of their effects, especially on the blood-currents through the different orifices.

Endocardial murmurs are not always due to lesions of structure or organic disease. They occur as a result of certain blood-changes and of functional disorder of the heart. The latter are distinguished as *inorganic murmurs*, while those dependent on structural changes are called *organic murmurs.* It is of great importance to discriminate, in practice, between organic and inorganic murmurs. With proper knowledge and care this can generally be done. The points involved in the discrimination are to be considered. I shall, however, first consider murmurs which it is assumed are organic, and afterwards point out the means of making the distinction in practice.

Inorganic murmurs will claim consideration also in connection with functional disorders of the heart. It is then to be understood that, for the present, reference is had to organic murmurs.

Organic murmurs, as regards their sensible characters, differ in a marked degree in different cases. In the majority of cases they resemble a bellows sound. Murmurs of this description are said to be *soft murmurs*. They vary greatly in intensity, pitch and duration. In some instances they are so feeble as to be just appreciable; in other instances they are so loud as to be heard over the whole chest, and are sometimes perceived by the patient, especially in the night time. In several cases that have come under my observation, patients have accurately described the sound, which had attracted their attention before any exploration of the chest had been made. It is sometimes heard by others at a distance from the chest. I have known instances where this has occurred to persons occupying the same bed with patients affected with disease of the heart. It may not be amiss to mention, in this connection, that physicians and medical students not unfrequently imagine they discover, during the night, a cardiac bellows murmur in their own persons, mistaking for it a sound produced by the movement of the head on the pillow synchronously with the ventricular systole, or, possibly, by the current of blood in the cerebral arteries. Between the extremes of feebleness and loudness, different cases present every degree of gradation as respects intensity. The pitch varies within certain limits. Bouillaud first proposed the plan of representing the pitch of bellows murmurs by whispered words and letters, which is much more convenient and clear than verbal descriptions. The highest pitch, unless the sound become musical, may be represented by the letter S, and a lower grade, but still acute, by the letter R. When the pitch is low it is often represented by the word WHO, and when still more grave by the word AWE. These letters and words were selected for this purpose by Dr. Hope. The pitch of the murmur is a point of some importance, but it has far less significance than was supposed by the distinguished author just named, whose labors contributed largely to our present knowledge of the diagnosis of diseases of the heart. The duration of bellows murmurs is by no means uniform in different cases. It is sometimes extremely brief, resembling the shortest possible puff, and in other cases prolonged over half, three-quarters and even a larger proportion of the heart's beat or revolution.

Murmurs which lack the softness of those just referred to, and

which bear but a remote resemblance, or none whatever, to a bellows sound, are distinguished as *harsh* or *rough murmurs*. They are always organic; in other words, they invariably denote structural lesions of some kind. These also differ greatly in their sensible characters. Different varieties have been described and named from their resemblance to certain sounds. Thus, French writers recognize *filing*, *grating*, and *rasping* murmurs (*bruit de lime*, *bruit de râpe*, *bruit de scie*, etc.), comparing the sounds to those produced by filing and rasping wood. In a case which recently came under my observation, the sound was precisely like the croaking of a frog.[1] The rough murmurs, in fact, in different cases suggest various comparisons. The varieties, however, are of very little practical consequence. They have not, severally, any special significance. The rationale of the diversity is not well understood. It suffices to consider them simply as presenting different modifications and degrees of roughness, the latter being the only distinctive feature worthy of being noted. The distinction, indeed, between roughness and softness is not of much importance in a practical point of view, beyond the fact that the former denotes, intrinsically, structural lesions. The absence of roughness, however, is by no means evidence that structural lesions do not exist. It is stated by some writers that a rough murmur indicates something more than the existence of structural lesions, viz: the presence of calcareous deposit on the valves, orifices, or lining membrane of the heart-cavities or vessels. This statement is not correct. I have met with cases in which the murmur was notably rough and no calcareous deposit was found after death. On the other hand, in cases in which the deposit is abundant, the murmur is frequently devoid of roughness. Rough murmurs, as a rule, are more intense than soft murmurs. They are oftener perceived by the patient. In a case in which the murmur resembled the croaking of a frog, the sound was accurately described by the patient, and was distinctly heard when the ear was in close proximity to the chest, but not in actual contact. The duration of rough as of soft murmurs varies considerably in different cases, but they are rarely, if ever, so brief as the latter in the instances in which a short puff only is heard. A soft murmur in some cases during the progress of disease is converted into a rough murmur; and the converse of this

[1] Following the custom of some French writers of considering a peculiar sound as a variety of murmur and giving it a name, this should be called *bruit de grenouille!*

also occurs. A murmur may be soft when the action of the heart is feeble or moderately strong, and become rough when the organ is excited into greater activity; and, conversely, it is possible that a murmur which is soft when the heart acts with violence, may become rough when the organ is more tranquil.

Murmurs sometimes have a musical intonation. The sounds are compared to the sibilant rale, the cooing of a dove, the whining of a puppy, etc. (*bruit de sifflement*, *bruit sibilant*, *bruit de roucoulement*, *de piaulement*, etc.). These are as much less frequent than the rough murmurs as the latter are more infrequent than soft murmurs. They are interesting only as clinical curiosities. They have no special pathological or diagnostic significance, except that they denote the existence of organic disease. They are preceded by, and may give place to, the common bellows murmur. They may alternate with the latter in different conditions of the heart's action. As remarked by Bouillaud and Hope, it is as intelligible that a bellows murmur may be transformed into a musical tone as that a change in the disposition of the lips changes a blowing into a whistling sound.

This will suffice for a general description of endocardial or valvular organic murmurs. Their relations to the two sounds of the heart and to the different currents of blood, the different situations to which they may be limited, or in which they are heard with their maximum intensity, and the directions in which they are transmitted, are of far greater importance, as regards diagnosis, than their intrinsic characters. To these practical points attention will now be directed.

The passage of the blood through the cavities and orifices of the heart and the large vessels, in health, takes place noiselessly, excepting the normal heart-sounds which have been considered.[1] The bulk of the heart, the capacity of its cavities, the smoothness of the endocardium, the size of the orifices and vessels, the protection afforded by the valves against regurgitating currents, and the quality of the blood, are all so nicely harmonized that the circulation is unattended by a murmur unless abnormal conditions of some kind exist. The morbid changes which may give rise to adventitious sounds are various. The presence of a murmur involves only the fact that there is something abnormal. It does not indicate the seat or nature of the change that has taken place, until certain contingent circumstances are taken into account. Of the

[1] Chap. I., page 58 *et seq.*

abnormal conditions which clinical observation has shown to be productive of murmurs, the more important are the following: Roughness of the endocardium and of the membrane lining the aorta or pulmonary artery; the presence of deposits (exudations and coagula) which interrupt or disturb the current of blood, and the projection into the current of rigid, unyielding valves; contraction of the auriculo-ventricular or the ventriculo-arterial orifices; dilatation and contraction of the large vessels connected with the heart; insufficiency of the valves designed to protect the orifices just named, due either to lesions affecting the valves or to dilatation of the orifices; aneurismal dilatation of a portion of one of the ventricles; dilatation of the whole ventricle, rendering the size of the cavity disproportionate to the quantity of blood; the formation of clots in the ventricles; certain alterations in the composition of the blood; sudden diminution in the circulating mass of blood; functional disorder of the action of the heart; communication between the two ventricles, and other congenital malformations. In this list of abnormal conditions, the alteration in the composition of the blood, sudden diminution in the circulating mass, and functional disorder of the heart, are to be distinguished as *inorganic*, that is, structural lesions of the heart are not involved. Murmurs due to these conditions are therefore *inorganic murmurs*. Murmurs dependent on any of the other conditions enumerated are *organic murmurs*.

The physical conditions on which, in the vast majority of cases, murmurs are dependent, are classed under the head of *valvular lesions*, which were described in the preceding chapter. Under this head are embraced the greater part of the conditions just enumerated. The morbid appearances which the valves and orifices present in different cases, it has been seen, are extremely diversified, consisting of various forms and degrees of roughness from calcareous deposits; vegetations of greater or less size, and more or less numerous, and variously disposed; thickening, on the one hand, and attenuation, rupture, perforations, and a cribriform condition, on the other hand; crumpling and contraction to a greater or less extent; cartilaginous stiffness and ossific rigidity; disruption of tendinous cords and fleshy columns; adhesion of the valves to each other and to the inner surface of the heart or vessels; congenital deficiency or deformity, etc. These changes, it has also been seen, are important or serious in proportion as they involve, *firstly*, contraction of one or more of the orifices and con-

sequent obstruction to the free passage of blood; *secondly*, insufficiency of the valves or patency of one or more of the orifices, and consequent regurgitation; *thirdly*, contraction and patency combined. These immediate effects give rise to those secondary and remote derangements of the circulation which are observed to result from valvular lesions, and eventuate in enlargement of the heart progressively increasing in proportion to the duration and amount of either obstruction or regurgitation, or both. But morbid conditions included under the head of valvular lesions may exist which involve neither contraction nor insufficiency, and, consequently, do not occasion either obstruction or regurgitation. Thickening, roughening, vegetations, etc., may be present without the results just mentioned, and therefore without the ulterior consequences in which consists the importance or seriousness of valvular lesions. Valvular lesions by no means necessarily involve immediate danger; they may exist for a long period and no evils arise from them. In a practical point of view, this is a fact of great importance when it is considered that innocuous lesions give rise to endocardial murmurs. It is to be borne in mind that valvular lesions which do not occasion any of the evil consequences arising from obstruction or regurgitation, nevertheless may involve physical conditions requisite for the production of murmurs. This important point will recur after the following questions have been considered: Are valvular lesions which do involve either obstruction or regurgitation, or both, uniformly or generally accompanied by murmur? What are the circumstances which, considered in connection with the presence of a murmur, enable the auscultator to determine whether existing lesions involve either contraction or insufficiency, or both? Can the particular seat of valvular lesions be determined, and, if so, in what manner? The practical importance of these questions is sufficiently obvious. Before proceeding to their consideration, the reader may be reminded of the fact that in the vast majority of the cases of valvular lesions they are confined to the left side of the heart, affecting the aortic or mitral orifices, singly or combined. The questions just propounded, therefore, will relate mainly to the valves and orifices connected with the left ventricle.

Are valvular lesions which involve either obstruction or regurgitation, or both, uniformly or generally accompanied by murmur?

This question may be answered affirmatively as regards the *general*, but not as regards the *uniform* occurrence of murmur. Clinical experience shows that in the vast majority of cases murmur coexists; and it is probable that in most of the instances in which at a certain stage in the progress of the disease careful physical exploration fails in discovering any murmur, it either has existed or becomes developed subsequently. Cases have been reported in which valvular lesions involving considerable and even great obstruction and regurgitation, were found after death, and no murmur had been discovered during life. Such cases are rare; several are given by Dr. Stokes.[1] The same author cites cases in which murmurs had existed, but disappeared in the progress of the disease, generally towards the close of life.[2]

These cases show that the physical conditions necessary for the production of a murmur are not constantly associated with even extensive lesions of the valves and orifices; and that when these conditions are associated (which is a rule with very few exceptions), a murmur sometimes disappears in consequence of diminished power of the ventricular contractions. Owing to the enfeebled action of the heart which often precedes, for a greater or less period, a fatal termination, a murmur which has existed may disappear, the blood not being propelled with force sufficient for its continuance, notwithstanding the persistence of physical conditions sufficient for its production. The practical bearing of the question under consideration relates chiefly to the value of the murmurs in a negative point of view; in other words, to the evidence afforded by the absence of murmur against the existence of valvular lesions. It follows from the statements just made, that the absence of murmur is not positive proof of the non-existence of serious valvular lesions. But it follows, also, that the probability of such lesions being present when a murmur is not discoverable, is exceedingly small; so small, indeed, that it may be almost said to be with safety disregarded in diagnosis, especially if those cases are excluded in

[1] Diseases of the Heart and Aorta, Am. ed., p. 157 *et seq.*

[2] Dr. Stokes refers to a series of cases illustrative of the disappearance of murmur in progressive valvular disease, collected by J. M. O'Ferrall, M. R. I. A., and published in the *Dublin Journal of Medical Science*, series 1st, vol. xxiii., 1843.

which an exploration of the chest is made when the action of the heart is weakened by the failure of the vital forces, or by any causes depressing the muscular power of the organ. It is a point of great importance to determine in individual cases whether valvular lesions do or do not exist. The presence of a murmur by no means warrants the conclusion that lesions do exist, as will appear more fully after inorganic murmurs have been considered. The absence of murmur, on the other hand, warrants the conclusion that lesions do not exist, the probability of error being exceedingly small, provided the heart be not from any cause greatly weakened.

What are the circumstances which, taken in connection with a murmur, enable the auscultator to determine whether existing lesions involve either obstruction or regurgitation, or both?

With reference to this question, as well as to that which follows, it is necessary to have a clear idea of the relations of endocardial murmurs to the two sounds of the heart, respectively, and to the different currents of blood. After the systolic contraction of the ventricles, the blood passes through the auriculo-ventricular orifices from the auricles into the ventricles. Limiting the attention to the left side of the heart, this may be designated the *direct mitral current.* The systolic ventricular contractions impel the blood from the cavity of the ventricle into the arterial vessels. The current of blood from the cavity of the left ventricle into the aorta, may be distinguished as the *direct aortic current.* These are the normal blood-currents. Others are incident to disease. If the mitral valves are insufficient, more or less of the blood contained in the cavity of the left ventricle is impelled backwards into the left auricle. Here, then, is a regurgitant current which does not exist when the valves are sound and sufficient. It may be called a *mitral regurgitant current.* If insufficiency of the aortic valves occurs as an effect of lesions in this situation, the blood which remains in the aorta after the ventricular systole, regurgitates into the ventricular cavity to a greater or less extent. This may be distinguished as an *aortic regurgitant current.* Now, each of these four currents may give rise to a murmur. Murmurs produced by these different currents may be named accordingly. Hence, there may be a *mitral direct murmur;* a *mitral regurgitant murmur;* an *aortic direct murmur*, and an *aortic regurgitant murmur.* These several murmurs sustain different

relations to the heart-sounds, as will be obvious on a little consideration.

A *mitral direct murmur* follows the diastolic or second sound of the heart, and precedes the systolic or first sound; in other words, it takes place during the long silence or pause which separates the diastolic and systolic sounds. If we divide the murmurs into two classes (which it is convenient to do), viz., into diastolic and systolic murmurs, according to their relations to one of the two heart-sounds, a mitral direct murmur will be included in the class of diastolic murmurs. In point of fact, it occurs just before the systolic sound, and is, strictly speaking, more accurately called a presystolic than a diastolic murmur. For convenience, however, it may be distinguished as the *mitral diastolic murmur.*

A *mitral regurgitant murmur*, on the other hand, being produced by the ventricular systole, accompanies or follows the systolic sound. It belongs, therefore, in the class of systolic murmurs, and may be called the *mitral systolic murmur.*

An *aortic direct murmur*, also produced by the ventricular systole, is a systolic murmur; it accompanies or follows the systolic sound, and may be called the *aortic systolic murmur.*

An *aortic regurgitant murmur*, on the other hand, produced by the retrograde current from the aorta into the ventricle after the systolic contraction, either follows or replaces the diastolic sound. It is, therefore, a diastolic murmur, and may be called the *aortic diastolic murmur.*

The following recapitulation shows, at a glance, the titles of the different murmurs and their relations, respectively, to the blood-currents and heart-sounds:—

Systolic murmurs, accompanying, replacing, or closely succeeding the systolic or first sound of the heart, consist of, 1st. A mitral regurgitant or a mitral systolic murmur; and, 2d. An aortic direct or an aortic systolic murmur.

Diastolic murmurs, accompanying, replacing, closely preceding or following the diastolic or second sound of the heart, consist of, 1st. A mitral direct or a mitral diastolic murmur; and, 2d. An aortic regurgitant or an aortic diastolic murmur. Each of these four murmurs claims distinct notice with reference to the important practical question under consideration.

1. *Mitral Direct or Diastolic Murmur.*—This occurs more unfrequently than the other three murmurs. It is rarely observed, not

because the physical conditions, so far as lesions are concerned, are proportionately infrequent, but in consequence of the contraction of the left auricle not taking place with power sufficient to impel the current of blood through the auriculo-ventricular orifice with force enough to give rise to audible sonorous vibrations. Some have disputed the possibility of this murmur. Not only does it occur, but there is reason to believe that its occurrence is less rare than is generally supposed. Roughness of the mitral valve on its auricular aspect, and especially irregular calcareous deposits, vegetations, etc., projecting from this surface of the valve, or from the orifice, rippling, as it were, the current of blood in its direct course from the auricle to the ventricle, are physical conditions which give rise to this murmur; but they are seldom adequate unless contraction of the mitral orifice be superadded. If there be sufficient obstruction at this orifice, the blood is thrown into sonorous vibrations in consequence of the greater velocity of the stream. Hypertrophy of the muscular portion of the left auricle contributes to the production of the murmur, or renders it more intense, the blood being impelled through the contracted orifice with greater force. As a rule, a mitral direct or diastolic murmur denotes not only the existence of mitral lesions, but mitral contraction. This rule, however, is not without exceptions, but they are probably extremely rare.

The existence of contraction and consequent obstruction is further shown by associated circumstances. One of these is intensification or reinforcement of the pulmonary second sound of the heart in the second left intercostal space. The significance of this sign has been alluded to in connection with the subject of hypertrophy of the right ventricle.[1] It will be noticed presently under the head of abnormal modifications of the heart-sounds incident to valvular lesions. Symptoms, in distinction from signs, are also to be taken into account. Those which, in connection with the presence of a mitral direct or diastolic murmur, point to mitral contraction, and which afford, measurably, evidence of the amount of obstruction, are phenomena denoting congestion of the lungs, viz., dyspnœa, defective oxygenation of the blood, hæmoptysis, and pulmonary apoplexy. The pathological relations of these events have been already considered.[2] The manner in which this murmur, as also the other murmurs, may be localized by auscultation,

[1] Chap. I., page 65.

[2] *Vide* Chap. III.

will be considered in connection with the question which follows that under present consideration.

2. *Mitral Regurgitant or Systolic Murmur.*—This is as common as the preceding murmur is rare. According to my own observations, it is the murmur most frequently met with in cases of organic disease of the heart. Whenever the mitral valve is rendered insufficient by abnormal changes, a portion of the blood contained in the left ventricle is driven backwards by the ventricular systole into the left auricle. This regurgitant current passes through an orifice frequently contracted and generally more or less irregular, the surfaces roughened by calcareous deposit, warty excrescences, etc. The physical conditions pertaining to the lesions are the same as in the cases in which a mitral direct or diastolic murmur is produced, with this important difference, viz., in the latter instance the force of the current is comparatively feeble, being due to the contractile movement of the left auricle, while in the former instance the blood is propelled with a momentum commensurate with the power of the left ventricle. The force of the regurgitant current, in fact, is such that a murmur is almost invariably produced; the exceptions are so few that, practically, the fact of their occurrence may be almost disregarded. Insufficiency of the mitral valve, then, is accompanied by a mitral regurgitant murmur in the vast majority of cases. Is it true, on the other hand, that a systolic murmur referable to the mitral orifice as uniformly denotes the existence of insufficiency or regurgitation? This question must be answered in the negative. There are rules, to be presently considered, which enable the auscultator to localize a systolic murmur at the mitral orifice. Now, of the instances in practice in which a murmur is referred to this situation, in only a certain proportion does regurgitation occur; in other words, a murmur may be produced at or near the mitral orifice, due to roughness, calcareous deposit, etc., without the valve being thereby rendered insufficient. It is important that this fact should be borne in mind. The gravity of valvular lesions, as has been seen, depends on the amount of obstruction and regurgitation resulting from them; hence, the importance of bearing in mind that a mitral systolic murmur is not always, strictly speaking, a regurgitant murmur, *i. e.*, the murmur may be produced without regurgitation. What, then, are the circumstances connected with the murmur which denote insufficiency or regurgitation? The diffusion of the murmur is an

important point with reference to this question. If the murmur be diffused over the left side, extending to the lateral surface of the chest, and, as is not unfrequently the case, even to the posterior surface, regurgitation may be assumed. This diffusion is more valuable in a positive than in a negative point of view; that is to say, regurgitation may exist without diffusion of the murmur, while the converse, as a rule, does not hold good. Other associated circumstances pointing to regurgitation are the symptoms and pathological events proceeding from pulmonary congestion, which have been considered in the preceding chapter. Considerable regurgitation, however, may continue for a long time before these become developed. Clinical observation shows that mitral regurgitation is long borne without serious inconvenience, especially if mitral contraction does not coexist. Symptoms, therefore, do not afford much aid in an early diagnosis of mitral insufficiency. A comparison of the aortic and the pulmonary second sound has an important bearing on the question just stated. Greater relative intensity of the pulmonic second sound is a sign of marked significance in this connection. It has a twofold significance. The intensity of the aortic second sound is diminished in proportion to the amount of blood which regurgitates through the mitral orifice, the stream which should pass into the aorta with each ventricular systole being lessened. The abnormal feebleness of the aortic second sound is thus proportionate to the degree of mitral insufficiency. In this point of view, the greater relative intensity of the pulmonic second sound is significant. But the intensity of this sound becomes positively augmented. When this is the case, it shows that the regurgitation has been sufficient to induce a degree of obstruction to the pulmonary circulation which has induced hypertrophy of the right ventricle in the manner already described. Attention to the diffusion of the murmur, and a comparison of the aortic and pulmonic second sound, enable the auscultator generally to determine with much positiveness whether a mitral systolic murmur denotes insufficiency, and also to form an idea of the amount of regurgitation. It may be added that the significance of a weakened aortic second sound is enhanced by evidence that the left ventricle is hypertrophied, inasmuch as, under these circumstances, the aortic second sound should be increased rather than diminished in intensity, provided the aortic valves are sound and mitral regurgitation does not take place.

Mitral insufficiency may exist either with or without mitral con-

traction, and, conversely, the latter may exist with or without the former. The morbid alterations which occur in this situation, however, involve insufficiency without contraction oftener than contraction without insufficiency, but both are not unfrequently combined. It follows from these facts that a mitral direct or diastolic murmur and a mitral regurgitant or systolic murmur may exist either separately or conjointly; that the existence of the former alone is extremely infrequent, while it is very common to meet with the latter by itself, and that the instances in which both are conjoined are more frequent than the instances in which the first exists separately. Clinical observation confirms the correctness of these conclusions.

A mitral regurgitant murmur was first recognized as such by Dr. Hope in 1825. It was first described by Dr. Elliottson in 1830.

3. *Aortic Direct or Systolic Murmur.*—In frequency of occurrence, this ranks next to the murmur last noticed. It is stated by some writers that it occurs oftener than either of the other murmurs. My own recorded observations do not lead to this conclusion. Of fifty-nine cases of valvular lesions in which either a mitral regurgitant or aortic direct murmur existed separately, the former was present in thirty-eight, and the latter in twenty-one. This murmur, assuming, for the present, that it proceeds from organic lesions, denotes a serious affection, or otherwise, according to the effect of the lesions as regards obstruction at or near the aortic orifice. The physical conditions necessary for the production of a murmur in this situation may exist without obstruction. Such instances are not very rare. There may be, under these circumstances, no immediate danger and no troubles referable to the heart affection. The physical conditions giving rise to the murmur may remain for an indefinite period innocuous. On the other hand, in proportion as obstruction to the aortic blood-current is involved, evils ensue, viz., accumulation of blood in the ventricular cavity; enlargement of the left ventricle by dilatation or hypertrophy, or commonly both; enlargement of the left auricle, pulmonary congestion, and the more remote consequences which are essentially those also resulting from obstructive and regurgitant lesions of the mitral orifice. There are no constant characters pertaining to the murmur itself which enable the auscultator to determine whether the lesions do or do not involve obstruction. Marked roughness or a musical intonation renders it highly probable that there is contrac-

tion at the orifice, due to expansion and rigidity of one or more segments of the valve or the presence of an abundant morbid deposit. But these conditions may exist without roughness of the murmur or a musical intonation; and, hence, the absence of the characters just named is not evidence against the existence of obstructive lesions. It is a point of importance to observe the aortic second sound of the heart. If this sound retain its normal intensity and purity, it shows that the aortic valve is competent to fulfil its function, a fact which warrants the exclusion of lesions affecting it sufficiently to give rise to obstruction. In a large proportion of the cases of obstructive lesions at the aortic orifice, the valve is involved sufficiently to compromise, to a greater or less extent, its function, and impair the intensity of the aortic second sound. This practical point is to be borne in mind. Aside from this, the evidence of the existence of obstruction, and also of its degree and duration, must be derived from the extent of enlargement of the left ventricle and the symptoms dependent on the remote effects of the heart affection. The cardiac enlargement, however, and the remote effects may proceed equally from aortic obstruction and aortic regurgitation, as in cases of mitral lesions they result alike from contraction and insufficiency. Enlargement of the heart accompanying valvular lesions, either at the mitral or aortic orifice, is, in general, proportionate to the amount and duration of obstruction or regurgitation, or both, which the lesions involve. The enlargement alone does not enable the diagnostician to discriminate between the obstructive and regurgitant lesions. As regards the localization of lesions at the mitral or aortic orifice, assistance is derived from this source, which will be referred to in connection with that subject.

4. *Aortic Regurgitant or Diastolic Murmur.*—This ranks next to a mitral direct or diastolic murmur as regards infrequency. It is more frequently met with than the latter, but much less frequently than the two other murmurs. An important fact which was stated in connection with the notice of the mitral regurgitant or systolic murmur, is equally true with respect to the murmur under present notice, viz: A murmur may be produced when no regurgitation takes place. Clinical experience has abundantly shown that in some instances a diastolic murmur referable to the aortic orifice exists, the aortic valve remaining sufficient. This occurs when, owing to roughness of the lining membrane of the aorta above the

valve, dilatation of the artery on the one hand, or contraction on the other hand, the retrograde movement of the column of blood, in this vessel, succeeding the ventricular systole, suffices to give rise to audible sonorous vibrations, although no blood falls backwards into the ventricle. The gravity of the lesions represented by a diastolic murmur referable to the aorta, depends mainly on the existence of insufficiency and the amount of regurgitation. What circumstances, then, associated with this murmur, enable the auscultator to decide whether regurgitation takes place or not? Attention to the aortic second sound of the heart is of importance with reference to this question. Has this sound its normal intensity and valvular quality unimpaired, the inference is that the valve is not to much extent, if at all, insufficient. Is the sound weakened and its normal quality not distinctly defined, this is evidence that the valve is involved so as to compromise its competency to fulfil its function, and consequently that regurgitation takes place. Is the sound extinguished, as is sometimes observed, either destruction or rigid expansion of the several segments of the valve has probably taken place. The degree to which the sound is impaired may be taken as, in a measure at least, a criterion of the amount of damage which the valve has received. Another point relating to this question is the diffusion of the murmur. If aortic regurgitation take place, the retrograde current of blood carries the murmur downwards so that it may be discovered over the body of the heart within the superficial cardiac region, and sometimes at the apex or even below the heart. This transmission rarely, if ever, takes place when a diastolic aortic murmur exists which is not regurgitant. Under these circumstances it may be propagated upwards even into the carotids, but to a limited extent, if at all, below the base of the heart.

An aortic direct or systolic murmur and an aortic regurgitant or diastolic murmur, may be present singly or combined. Regurgitation may take place without murmur, even when the physical conditions exist, owing to the force of the retrograde current being too feeble to occasion sonorous vibrations. Absence of an aortic regurgitant murmur, therefore, is not positive proof that there is no regurgitation. In the majority of the cases in which the lesions of the aortic valve are such as to give rise to a regurgitant murmur, the conditions for the production of a direct or systolic murmur co-exist. The aortic direct and aortic regurgitant murmurs, conse-

quently, are associated oftener than the latter is observed disconnected from the former.

The two murmurs produced at each of the two orifices, viz., the mitral and aortic, are not very infrequently presented in combination. The aortic and mitral murmurs may also be associated in the same case. Instances of the union of a mitral and an aortic murmur are sufficiently common. I have frequently met with three distinct co-existing murmurs; and it is quite possible for all four to be simultaneously present and distinguishable. It is conceivable, indeed, that eight distinct murmurs may be combined in the same case, tricuspid and pulmonic murmurs corresponding to those produced in the left side of the heart, being present. The latter murmurs are so extremely rare that it is not necessary to consider them in this connection. We come now to the consideration of the means by which the different murmurs, respectively, are localized and discriminated from each other.

Can the particular seat of valvular lesions be determined, and, if so, in what manner?

The first part of this inquiry has been already answered. It has been stated that it is practicable, generally, to localize valvular lesions. The mode in which this may be done is now to be considered. To refer a murmur to a particular valve or orifice seems to one unacquainted with the subject, to be a refinement in diagnosis not only difficult, but invested with an air of mystery. The rules, however, are extremely simple; their application is by no means intricate, nor does it require the exercise of any extraordinary skill or tact. The points involved in determining the particular seat of lesions, relate, 1st. To their relations to the heart-sounds; 2d. To the different situations in which murmurs are found to be most intense, and the different directions in which they are farthest propagated; 3d. To the pitch and quality of the murmur; and, 4th. To the condition of the heart-sounds considered in connection with the murmurs. In treating of this branch of the subject, it will be most convenient to consider the murmurs as embraced in two classes, viz: *systolic* and *diastolic*, *i. e.*, accompanying either the first or the second of the heart-sounds. The reader, however, will continue to bear in mind that a systolic murmur may be either an aortic direct, or a mitral regurgitant murmur; and that a diastolic murmur may be either a mitral direct, or an aortic

regurgitant murmur. These names, derived from the relations of the murmurs to the different blood-currents which have been described, are extremely useful in fixing these relations in the memory.

Localization of Systolic Murmurs.—In tracing an endocardial murmur to its source, the first point is to ascertain whether it be a systolic or a diastolic murmur. Generally this is unattended with difficulty; but in some instances it is not easy. The difficulty arises from the rapidity of the heart's action. If the heart sounds recur with great frequency, the systolic and diastolic sounds are not readily distinguishable from each other. The two sounds follow each other so quickly that the difference in duration between the two pauses or intervals is scarcely apparent. Moreover, under these circumstances, the first sound frequently loses its distinctive characters as regards its relative length, quality, and even pitch, and the two sounds become nearly or quite identical. Occasionally the two sounds cannot be discriminated, until the frequency of the ventricular contractions diminishes, or is reduced by certain sedative remedies, such as digitalis, or the veratrum viride. When the two sounds are with difficulty distinguishable from each other, it is, of course, proportionately difficult to determine which of the sounds an existing murmur accompanies. This difficulty, happily, is experienced in only a small proportion of cases. In the few instances in which a murmur cannot be referred to either sound, the localization of lesions is thereby made with less ease and positiveness. The rules, however, are still measurably available. Whenever the rapid action of the heart does not give rise to difficulty, the different characters as regards length, quality and pitch, which belong to the first or systolic sound, enable the auscultator to distinguish it with readiness. These characters have been already described.[1] If any doubt arise, with the finger upon the pulse of the patient while auscultation is practised, it is easy to determine with which of the two sounds the pulse is synchronous, or nearly so. The sound which occurs synchronously with the pulse is, of course, the systolic sound.

Assuming that a murmur has been ascertained to be systolic, it may be either a mitral regurgitant or an aortic direct murmur. The question which next arises is, how is it to be traced to the mitral or to the aortic orifice? If it be mitral, its maximum of intensity is at or near the apex of the heart. In some instances it

[1] Chapter I.

is most intense at the point where the apex-beat is seen, felt, or determined by auscultation. In other instances the intensity is greatest at a little distance to the left of the point of apex-beat. When the latter is the case, the explanation is probably that given by Dr. Sibson, viz: the murmur is somewhat obscured directly over the apex, by the intensity of the first sound, and sometimes by tinnitus.[1] The murmur may be confined within a circumscribed space around the apex. It is generally heard over the body of the heart, within the superficial cardiac region, but with diminished intensity. Above the base of the heart it is often feeble or wanting. It is not propagated into the carotids. If it be transmitted to the upper part of the chest, as is sometimes observed when it is unusually loud, the intensity is much less than over the body and apex, or below the heart. It is often diffused over the left lateral surface of the chest, and may extend to the posterior surface on the left, and sometimes on the right side. When heard on the back, its intensity is greater below than above the spinous ridge of the scapula, the maximum being generally near the lower angle of the scapula. The quality of the murmur may be soft or rough. It is soft in the great majority of cases. Roughness, however, belongs almost exclusively to systolic murmurs. Diastolic murmurs, at least, much more rarely than the systolic, present this quality. It very rarely has a musical intonation in this situation. The pitch varies in different cases, but, as a rule, is neither extremely high nor low. It is rarely as high as the letter R whispered, and still more rarely as high as S. It is seldom as low as AWE. In the larger number of instances, it is represented by WHO. The mitral valvular element of the first of the heart-sounds is frequently diminished or wanting, leaving the element of impulsion unduly predominant or solely present.[2] The diminution of this element is in proportion to the injury of the valve which the lesions have occasioned, and its absence shows that the valve is nearly or quite useless. The aortic second sound is diminished in intensity in proportion to the amount of blood which regurgitates through the mitral orifice. The pulmonic second sound is thereby rendered relatively more intense, and its intensity is often positively augmented by obstruction to the pulmonary circulation and hypertrophy of the right ventricle.

[1] Medical Anatomy.

[2] For an account of the two elements of the first or systolic sound, and the manner of exploring for the mitral and tricuspid valvular elements of this sound, the reader is referred to Chapter I. page 60, *et seq.*

The foregoing points distinguish a mitral regurgitant systolic murmur. If, on the other hand, a systolic murmur be an aortic direct murmur, its maximum of intensity is at or above the base of the heart. Its intensity is less over the body of the heart, within the superficial cardiac region, than at the base, and it may be lost in the latter situation. It is still more feeble and is often lost over the apex; and it is very rarely propagated below this point. The particular situation where it is most intense, is usually in the second or third intercostal spaces nigh to the sternum. In the third intercostal space on the left side nigh to the sternum the intensity is, in general, notably less than at the corresponding point on the right side. From the base of the heart it is propagated upwards for a greater or less distance, more so on the right than on the left side. It is often pretty loud at the sternal notch. It is heard in the neck over the carotids. It is sometimes heard on the posterior surface of the chest, and when this is the case its maximum is in the left interscapular space on a level with the spinous ridge of the scapula. It is soon lost below this point. The murmur may be soft or rough, the latter quality being much less frequent than the former. It is, however, oftener rough than a systolic mitral regurgitant murmur. The pitch is usually higher than in the majority of the instances of a mitral regurgitant murmur, often being represented by the letter R, whispered. The pitch, however, varies considerably in different cases. It has a musical intonation oftener than a mitral regurgitant murmur. The aortic second sound of the heart is frequently impaired and may be extinguished, the pulmonic second sound remaining.[1] The extent to which this sound is compromised, will, of course, correspond to the amount of injury to the aortic valves incident to the lesions which give rise to the murmur.

As already stated, the two systolic murmurs may be associated in the same case. This fact can generally be determined. The murmurs are rarely identical in quality and pitch; and each murmur has its maximum of intensity in different situations, and conforms to the characters which distinguish, on the one hand, a mitral regurgitant, and on the other hand, an aortic direct murmur. As a rule, the two murmurs in the same case may be localized with as

[1] The situations in which the aortic and pulmonic second sounds may be studied separately, are the second intercostal spaces on the right and left side of the sternum. See Chapter I. page 59.

much precision, as when either is present without the other in different cases.

Localization of Diastolic Murmurs.—A murmur having been ascertained to be diastolic, *i. e.*, sustaining a closer relation to the second than to the first sound of the heart, the question to be then settled is, whether it be a mitral direct murmur or an aortic regurgitant murmur. The points involved in this discrimination are less strongly marked than in the case of the systolic murmurs. But in most instances the distinction can be made with proper knowledge and care.

A mitral direct murmur, as before stated, is strictly speaking pre-systolic. It occurs just before the first or systolic sound, and may be continued into that sound. This is due to the fact that the contraction of the left auricle precedes, by a very short interval, the contraction of the left ventricle, the latter appearing to be a continuation of the former. This is apparent on examining the movements of the heart exposed to view in vivisections practised on animals of large size, and in cases of ectopia. The murmur is usually accompanied by a systolic mitral regurgitant murmur, the same lesions giving rise to both. Its maximum of intensity is over the body or apex of the heart. It is rarely diffused in any direction without the præcordia. It is feeble or not distinguishable at the base of the heart. The murmur is rarely intense, and in the vast majority of cases its quality is soft. The pitch is usually low. It may be represented by the whispered word AWE. The mitral valvular element of the first sound may be more or less impaired, but the aortic second sound (assuming that aortic lesions do not co-exist) preserves its normal intensity. The latter is an important point in discriminating this murmur from an aortic regurgitant murmur. The pulmonic second sound is not only relatively more intense than the aortic in consequence of the diminished intensity of the latter, but is positively intensified, mitral contraction generally existing when this murmur is present, and, as a consequence, pulmonary congestion and hypertrophy of the right ventricle.

If the diastolic murmur be an aortic regurgitant murmur, it either replaces or follows more closely upon the second sound than a direct mitral murmur. It is due to the same force which causes the aortic second sound, viz., the recoil of the elastic coat of the aorta, while the mitral direct murmur is produced by the contraction of the left auricle. The former occurs prior to the

latter, and hence the corresponding murmur takes precedence in point of time. The intensity of this murmur is not, as is stated by some writers, greatest at the base of the heart, but below, over the body of the organ, nigh to the left margin of the sternum. It is, however, more likely to be heard at the base, in the second intercostal space on the right side of the sternum, than a mitral direct murmur, and this is an important point of distinction. It is generally feeble and very rarely rough in quality. The pitch is usually low. In most instances it is associated with a systolic aortic direct murmur, the same lesions giving rise to both murmurs. The aortic second sound is usually more or less impaired, and in some instances is extinguished. A notable diminution of the intensity of this sound or its extinction, provided neither mitral contraction nor regurgitation co-exists, is proof positive that the diastolic murmur has its source in aortic regurgitation. On the other hand, the integrity of the aortic sound and a diminished intensity of the mitral valvular element of the first sound, constitute hardly less proof that a diastolic murmur is referable to the mitral orifice. Intensification of the pulmonic second sound occurs as a more remote and less constant result than in connection with the lesions which give rise to a mitral direct murmur.

The two diastolic murmurs may be associated in the same case, but instances of this combination are vastly less frequent than the union of the systolic murmurs. It is, however, as already stated, very common for one or both of the systolic murmurs to be associated with a diastolic murmur. Examples of three murmurs, two systolic and one diastolic, are not very infrequent. Usually, each may be referred to its source. It is not claimed that the rules of localization which have been briefly pointed out are infallible. Exceptional instances will occur in which the source of a murmur is uncertain. But in the great majority of cases it is determinable without much doubt or difficulty. This statement is based on considerable practical experience, and on the analysis of a large number of recorded cases.

To assist the reader in fixing in the memory the points involved in the localization, which have just been considered, the following tabular view is added:—

RECAPITULATION OF POINTS INVOLVED IN THE LOCALIZATION OF SYSTOLIC AND DIASTOLIC MURMURS.

SYSTOLIC MURMURS.

Mitral Regurgitant.	Aortic Direct.
Maximum of intensity at or near the apex of the heart. Comparatively feeble or wanting at the base.	Maximum of intensity at or above the base of the heart in the second or third intercostal space, near the sternum. Intensity notably diminished over body of heart and at the apex.
Not propagated above the base of the heart. Not heard over carotids.	Propagated above the base of the heart, and often heard over carotids.
Often diffused over left lateral surface of chest.	Rarely heard over left lateral surface of chest.
If heard in the interscapular space, most intense near the lower angle of scapula.	If heard in the interscapular space, most intense as high as the spinous ridge of scapula.
Aortic second sound weakened but distinct.	Aortic second sound often more or less indistinct.
Pulmonic second sound intensified.	Pulmonic second sound less frequently intensified.
Mitral valvular element of the first sound more or less impaired.	Mitral valvular element of the first sound not impaired.

The murmur, in a certain proportion of cases, rough, but, in the majority of instances, soft.

The pitch varying, in the majority of cases, between the sounds represented by the letter R and the syllable WHO, whispered.

DIASTOLIC MURMURS.

Mitral Direct.	Aortic Regurgitant.
Occurs just before the systolic or first sound.	Replaces or follows closely the diastolic or second sound.
Often associated with a systolic mitral regurgitant murmur.	Often associated with a systolic aortic direct murmur.
Maximum of intensity over body or apex of heart.	Maximum of intensity over body of heart, near the sternum.
Feeble and often not appreciable at the base of the heart.	Generally appreciable at the base of the heart.
Mitral valvular element of first sound may be impaired.	Mitral valvular element of first sound not impaired.
Aortic second sound may be normal.	Aortic second sound often impaired and sometimes extinguished.
Pulmonic second sound intensified.	Pulmonic second sound less frequently intensified.

Always feeble in comparison with the intensity which systolic murmurs often present.

The quality almost invariably soft.

The pitch generally lower than systolic murmurs; often represented by the whispered word AWE.

Rarely diffused without the præcordial region.

Pathological Import of Organic Endocardial Murmurs.

It is highly important for the student to form a just notion of the extent to which organic murmurs are available in furnishing information respecting pathological conditions. The considerations which have been presented have related mainly to diagnosis. They show that organic lesions are accompanied by an organic murmur in the great majority of cases; and, conversely, the absence of murmur renders it almost certain that organic lesions do not exist. So far, the practical value of auscultation in this application is very great. Further than this, valvular lesions may generally be localized at one or more of the orifices of the heart by attention to certain points pertaining to the murmurs. Moreover, the study of the murmurs enables the auscultator often to determine whether obstruction or regurgitation, or both, at one or more of the orifices, are consequent on existing lesions. These ends are sufficiently important to render invaluable the aid thus derived from auscultation. The information, however, derived from the murmurs is limited mainly to these ends. The character of the structural changes which have taken place, and the amount of damage which they have occasioned, are to be determined by other means than the study of the murmurs. In a practical view, it is far more important to establish these points than the mere existence of organic lesions of some kind, or their particular situation and immediate effects on the blood-currents. In determining these points, the heart-sounds are to be studied with reference to abnormal modifications, or otherwise, and the existence and extent of enlargement of the heart are to be ascertained. The means of ascertaining the existence of cardiac enlargement and measuring its extent have been fully considered in a former chapter. Abnormal modifications of the heart-sounds, in connection with endocardial murmurs, have also been referred to. The latter will be again noticed presently under a distinct head.

It is to be borne in mind that lesions which are innocuous as regards any immediate effects, *i. e.*, which do not occasion either obstruction or regurgitation, may give rise to murmurs. Fibrinous

or calcareous deposits on the valves, orifices, or within the aorta, without valvular insufficiency, contraction or dilatation, may supply the physical conditions for audible sonorous vibrations. Dilatation of the cavities of the heart is said sometimes to give rise to a murmur, which has been attributed to a disproportion between the size of the cavities, and the quantity of circulating blood. Without denying the possibility of the production of a murmur under these circumstances, it is certainly an event of extremely rare occurrence. Dilatation without over-distension is an anomaly which rarely occurs. A more rational explanation is to attribute the murmur, when produced, to an abnormal condition of the blood, an explanation which brings the murmur into the class of inorganic murmurs. Serious lesions are by no means to be predicated on the existence of a murmur which is undoubtedly of organic origin. This is the practical precept to be enforced. Nor is the intensity of a murmur to be taken as any criterion of the importance of the lesions which give rise to it. An intense murmur may accompany trivial lesions, and, on the other hand, the most serious lesions may give rise to a feeble murmur. Indeed, as remarked by Dr. Walshe, feebleness of the murmur may, in some instances, constitute evidence of the gravity of lesions, showing that the heart has become weakened by the dilatation and over-distension of its cavity incident to the lesions. Roughness of a murmur also, it is to be recollected, is no guide to the nature or extent of the structural changes which it indicates. The same remark is applicable to the diffusion of a murmur, when it is aortic. Diffusion of a mitral regurgitant murmur, on the other hand, without the præcordial region, may, in general, be considered as denoting regurgitation; but it is in nowise a criterion of the amount of regurgitation, or, in other words, valvular insufficiency.

An organic murmur is in some instances neither propagated above the heart, nor diffused in any direction beyond the heart, but confined within the limits of the præcordial region. The lesions giving rise to the murmur in these instances are within the left ventricle, and may be situated either on the ventricular aspect of either the mitral or aortic valve. It is difficult or impossible to localize the lesions under these circumstances. For an example, in a case in which vegetations of considerable size were dependent from the inferior surface of the aortic valve, the valve not being otherwise affected, the murmur was confined to the region of superficial cardiac dulness, its maximum of intensity not being marked at any point within this space. Murmurs of this description may

be distinguished as *intra-ventricular murmurs*,[1] and the inference to be drawn from them is that the valves are not affected to the extent of interrupting materially their functions.

In treating of the subject of endocardial organic murmurs in this chapter, reference has been had, for the most part, to those occurring in connection with chronic valvular affections of the heart. Organic murmurs, as will be seen hereafter, also occur in connection with heart-clots, in cases of congenital malformations, and their production becomes an important physical sign of the development of endocarditis. Without due attention, murmurs taking place within an aortic aneurism situated near the heart, are liable to be mistaken for endocardial murmurs.

Inorganic Murmurs.

As already defined, a murmur is inorganic when it is produced independently of organic or structural lesions. An endocardial murmur may be present when there are no lesions. The practical importance of being able to determine whether an existing murmur be organic or inorganic is sufficiently obvious. This discrimination, happily, can be made in practice in the great majority of cases. The points involved in the discrimination claim attention in this connection.

An inorganic murmur, as a rule, proceeds from an abnormal change in the composition and properties of the blood. The precise nature of the change is perhaps not positively ascertained. At all events, a discussion of this subject need not be here introduced. Whatever be the requisite conditions, they occur in a certain proportion of cases of anæmia and chlorosis. The murmur in these instances is said to be of *hæmic* origin. It was observed by Marshall Hall, in his researches on the effects of the loss of blood, that the sudden abstraction of a large quantity of blood led to the development of a transient bellows-murmur. Other observers have verified this fact. It is occasionally observed under circumstances which seem to render it probable that it proceeds from deficient or irregular contraction of the papillary muscles, involving temporary

[1] *Intra-ventricular*, in distinction from murmurs produced at the orifices and propagated for a greater or less distance beyond the heart, either above the base, if the lesions are aortic, or to the left of the heart if the lesions are mitral and involve insufficiency.

insufficiency and regurgitation. Its occurrence in some cases of chorea has been accounted for in this way. Thus produced, the murmur is said to be of dynamic origin. It is produced in some persons in health by the violent action of the heart which follows active muscular exertion, disappearing when the organ resumes its usual tranquillity. It is occasionally observed in the course of a variety of affections, when post-mortem examinations in cases of death, and its disappearance, leaving no signs or symptoms of cardiac disease, after recovery, show that it does not proceed from organic causes. The continued and eruptive fevers, uræmia, and hysteria, are among the affections in which it sometimes occurs. Its occurrence is not infrequent during pregnancy. What are the characters which distinguish these murmurs from those of organic origin?

Inorganic murmurs are uniformly systolic, *i. e.*, they accompany only the first of the heart-sounds. Diastolic murmurs are always of organic origin.

In the vast majority of cases, inorganic murmurs are heard at the base of the heart, and are not propagated far above, and, more especially, not below this point. They are very rarely heard at the apex, but if propagated to the apex, their maximum is at the base. This, at least, is true of all cases of inorganic murmurs of *hæmic* origin. It is only the very rare and somewhat dubious instances of murmurs of *dynamic* origin, that are produced at the auriculo-ventricular orifices, and, consequently, heard at the apex. These are characterized by temporary duration or intermittency. Hence, it may be stated that, as a rule, every persistent, constant murmur referable to the mitral orifice, denotes organic lesion of some kind, and, as a rule, organic murmurs are constant and persistent, while inorganic murmurs, wherever produced, are fluctuating and variable, being sometimes discoverable only when the body is in a certain position.

An inorganic murmur is uniformly soft. If this rule be not invariable, the exceptional instances are exceedingly infrequent, and the roughness in exceptional instances is not marked, nor constant, occurring only when the action of the heart is unusually excited. Roughness, therefore, may be considered as evidence that the murmur is organic. This statement will apply equally to an endocardial musical murmur. An inorganic murmur is always feeble. Intensity is evidence of organic origin.

An inorganic murmur may be produced either at the aortic or pulmonic orifice, or simultaneously at both orifices. If it be pul-

monic, as shown by its being either limited to, or having its maximum of intensity in the left second or third intercostal space, it is probably inorganic, in view of the great infrequency of lesions situated at this orifice. Congenital malformations are to be excluded from this statement, for these are more liable to affect the pulmonic than the aortic orifice. In this connection it may be mentioned that pressure with the stethoscope in these intercostal spaces over the pulmonic artery, will sometimes develop a bellows murmur in that vessel. This is observed in young persons whose costal cartilages are flexible. The murmur is due to pressure on the artery, as in the case of other arteries, more accessible, such as the carotid, iliac, femoral, etc. It is well known that light pressure on these arteries frequently develops a bellows murmur.

Inorganic murmurs occur in anæmic persons, and the palpable indications of anæmia are generally manifest. The coexistence of anæmia is a point to be considered in the discrimination. This condition, it is true, may coexist with valvular lesions, and contribute to render more intense and diffused the murmurs due to the latter. Anæmia alone by no means warrants a conclusion that a murmur is inorganic, but, added to other evidence, it strengthens this conclusion.

Concurrent bellows murmurs emanating from the large arterial trunks, the subclavian, carotids, etc., not due to pressure with the stethoscope, are evidence that an endocardial murmur is inorganic. This evidence is by no means complete in itself, but adds weight to that derived from other sources. A continuous murmur or hum produced in the jugular veins is very frequently associated with an endocardial murmur of hæmic origin. This venous hum, called, after Bouillaud, by the French writers, *bruit de diable* (from its resemblance to the sound of the humming-top, which is known popularly in France as *le diable*), has heretofore given rise to considerable discussion as regards its source. Laennec, who first observed it, referred it to the arteries. In this he has been followed by most French writers. Its origin in the veins was first demonstrated by Dr. Ogier Ward. It is a sufficient demonstration of the correctness of the latter view that the murmur is invariably suspended by interrupting the circulation through the veins, the arterial circulation continuing. The murmur is a continuous humming sound, having frequently a musical intonation. It is best heard over the jugulars, just above the clavicles, the patient being in the sitting or standing posture. It is highly characteristic

of anæmia, and its presence in conjunction with an endocardial murmur suspected to be inorganic, gives strength to this suspicion. Venous hum and arterial inorganic murmurs are not infrequently combined.[1]

Inorganic endocardial murmurs are much oftener observed in females than in males, a fact probably due to the greater frequency of anæmia in the former. Sex, therefore, is entitled to some weight in determining whether a murmur be organic or inorganic.

The heart-sounds, in connection with inorganic murmurs, retain their normal intensity and characters, or, if affected at all, their intensity is augmented; whereas, in connection with organic murmurs, they often present abnormal modifications, which are to be presently considered.

Finally, organic murmurs, in the great majority of the cases of chronic disease, when these first come under the cognizance of the physician, are associated with more or less cardiac enlargement. This is owing to the fact that valvular lesions do not, as a rule, occasion much inconvenience until they have induced enlargement of the heart. A murmur, under these circumstances, may have existed for many months or years, and escaped observation because the patient has never presented himself for examination. Coexisting enlargement, then, alone, renders it altogether probable that an endocardial murmur proceeds from organic lesions. It is true that enlargement of the heart, uncomplicated by valvular disease, may be associated with inorganic murmurs, but it is evident that this coincidence must be rare when it is considered that enlargement without lesions of the valves is by no means frequent. If, in connection with cardiac enlargement, a murmur be either mitral regurgitant or diastolic, it is certainly organic. Doubt can only arise when the murmur is an aortic direct murmur. On the other hand, in the vast majority of the cases in which a murmur is inorganic, the heart is not enlarged, a fact which can be positively determined by means of physical exploration.

With due attention to the several points which have been briefly considered, the auscultator need not be at a loss, in most instances, in discriminating with positiveness between organic and inorganic endocardial murmurs.

[1] Dr. Walshe remarks, with reference to the coexistence of an endocardial murmur and venous hum: "I do not remember ever to have observed an intra-cardiac spanæmic murmur unattended with venous hum."—*On Diseases of the Heart and Lungs*, second London edition, p. 242.

Abnormal Modifications of the Heart-Sounds in Cases of Valvular Lesions.

The study of the murmurs has so much engrossed the attention of clinical observers of late years, that the heart-sounds have not received that attention which their importance claims. Abnormal modifications of the heart-sounds afford, in certain cases, as has been seen, valuable aid in the localization of murmurs. They also serve to supply, in some measure, information, which, in a pathological point of view, is far more important than to determine the existence, situation and character of lesions, viz: respecting the amount of damage which the valves have sustained. The important practical points pertaining to these two objects have been already incidentally noticed, but it will not be amiss to recapitulate them under a distinct heading.

The results of the clinical study of the heart-sounds in health, show that the second or diastolic sound, consisting solely of a valvular element, is in fact composed of an aortic sound and a pulmonic sound, which are generally distinguishable from each other when the stethoscope is applied in the second intercostal space near the sternum on the two sides successively, the aortic second sound being heard on the right, and the pulmonic second sound on the left side. The first sound of the heart differs from the second, in being compounded of a valvular element and an element of impulsion. The valvular element, however, like the second sound, is composed of a mitral and a tricuspid valvular sound, which are distinguishable from each other when auscultation is practised successively in different situations. For further details the reader is referred to Chapter first, where this subject is fully considered.[1]

The abnormal modifications of the first sound may affect, either separately or conjointly, the two elements into which this sound is resolvable, and the two subdivisions of the valvular element of the sound; and the aortic and pulmonic sounds which make up the second sound of the heart, may also be affected singly as well as combined. It is in connection with valvular lesions more especially, that the different elements and their subdivisions are liable to be modified separately.

Mitral lesions impair the mitral portion of the valvular element of the first or systolic sound, other things being equal, in propor-

[1] Vide page 58, *et seq.*

tion to the extent of injury of the mitral valve which the lesions have occasioned. To isolate the sound referable to the play of the mitral valve, the stethoscope is to be placed without the left nipple at a distance sufficiently removed to eliminate the element of impulsion of the first sound. If the mitral valvular sound be abnormally feeble or wanting, provided the heart acts with sufficient vigor, it shows considerable or great imperfection in the action of the valve; and, conversely, if the sound preserve its normal intensity and quality, it may be inferred that, notwithstanding the existence of lesions, the valve is not, as yet, much damaged. A mitral regurgitant murmur, or a mitral direct murmur, either or both, co-exist in both cases; in the former case the murmur or murmurs may be feeble, and in the latter intense, the intensity of the murmur bearing no proportion to the gravity of the lesions. In cases in which the mitral valvular sound is notably impaired or extinguished, owing to the extent of injury to the valve, the tricuspid valvular sound may generally be distinguished by applying the stethoscope at or a little without the inferior or right border of the heart. Mitral regurgitation, in fact, leads to augmented intensity of the tricuspid sound by inducing hypertrophy of the right ventricle in the manner already described.

Mitral lesions involving obstruction or regurgitation, more especially the former, lead to diminished intensity of the aortic second sound, and an augmented intensity of the pulmonic second sound. The former is due to the column of blood propelled through the aorta by the ventricular contraction, being lessened either by the deduction of the quantity of blood which regurgitates, or by the deficient supply from the auricle to the ventricle. The latter proceeds from hypertrophy of the right ventricle. Both effects combine to render intensification or reinforcement of the pulmonic second sound, a valuable sign of mitral obstruction or regurgitation, as was first pointed out by Prof. Skoda.

In connection with the presence of a diastolic murmur, the normal intensity and quality of the aortic second sound constitute evidence that the murmur has its source at the mitral orifice. On the other hand, if, in connection with a diastolic murmur, the aortic second sound is notably impaired or extinguished, this goes to show that the murmur emanates from the aortic orifice.

Aortic lesions affect the aortic second sound, other things being equal, in proportion to the extent of injury of the valve of the aorta. If the play of this valve be defective, the sound loses more or less

of its normal intensity. The sound is extinguished when the valve is rendered useless or destroyed by disease. Instances of extinction of the aortic second sound are not very infrequent. In such instances the continuance of the pulmonic second sound shows that the loss of the aortic sound is not due to weakened action of the heart. An aortic direct or an aortic regurgitant murmur, or both, are present, indicating the fact of aortic disease, but the intensity and quality of the murmurs here, as in mitral lesions, do not constitute any criterion of the amount of damage to the valve. The abnormal modifications of aortic sound, however, afford definite information with respect to that important point.[1]

Purring Tremor.—This term is applied to a sense of vibration or thrill felt on placing the fingers or the hand on the præcordia. It is synonymous with the name applied to it by Laennec, viz: *frémissement cataire*, so called because it resembles the sensation communicated to the hand by the purring of a cat. Bouillaud compares it to the sensation felt when the hand is applied over the larynx of a person singing. These comparisons cause it to be easily recognized when met with for the first time. It is doubtless due to tremulous movements of the heart, which are propagated to the portion of the thoracic walls with which the heart is in contact.

Well-marked purring tremor may be considered as a sign denoting valvular lesions associated with hypertrophic enlargement of the left ventricle. If it occur under other circumstances, the instances are so infrequent that, for all practical purposes, the rule may be taken as invariable. The valvular lesions which most frequently give rise to it, are those of the mitral orifice permitting free regurgitation. A regurgitant current driven through this orifice with an abnormal force in consequence of the augmented muscular power of the ventricle, appears to be the immediate cause in the majority of instances. It accompanies or follows the ventricular systole, and is therefore synchronous with the first sound, the apex-beat and the pulse. A diastolic tremor must be exceedingly rare, but is stated by some clinical observers to occur occasionally. When due to mitral lesions, the tremor is felt within the superficial cardiac region below the level of the nipple. It may be more or less marked, the intensity depending, in a great measure,

[1] For a fuller consideration of this subject than is contained in this work, the reader is referred to the essay, by the writer, contained in the *Transactions of the American Medical Association*, vol. xi. p. 805.

on the power with which the left ventricle contracts. It may be present or strongly marked when the action of the heart is excited by any cause, and absent or comparatively feeble when the organ is tranquil. In the progress of disease, it diminishes and ceases as the heart becomes weakened. Although it has a pathognomonic significance when present, its absence is in nowise evidence against the existence of organic lesions, for it is wanting in a large proportion of the cases in which lesions exist.

The sign does not belong exclusively to mitral lesions. It accompanies, in some instances, aortic lesions associated with hypertrophy of the left ventricle. It is then felt nearer the base of the organ. A thrill due to the current of blood in the aorta is sometimes perceived above the heart in the second intercostal space on the right side. Vascular thrill in the large arteries which approach the surface, is sufficiently common in cases of anæmia. With the aneurismal thrill all observers are familiar.

Cardiac tremor is not a sign of much practical value in view of the fact that it is present only in a small proportion of cases of valvular lesions, and since other physical signs, which are constant, are readily available for diagnosis. It is, however, of sufficient interest and importance to be kept in mind in exploring the chest for the physical evidence of cardiac disease.

Purring tremor is to be distinguished from the tactile fremitus incident to the presence of solid deposit on the pericardial surfaces. The latter, which will be noticed in connection with pericarditis, is always accompanied by an exocardial murmur or friction sound on auscultation. Purring tremor, on the other hand, is almost invariably associated with one or more endocardial murmurs.

Diagnostic Characters of Lesions affecting the Mitral, Aortic, Tricuspid, and Pulmonic Valves or Orifices.

The diagnosis of valvular lesions is based on physical signs, together with the symptoms and pathological effects. These have been considered. The significance and diagnostic value of the different signs, symptoms, and effects, have been already pointed out. It remains to group together the more important of the characters which pertain to the different lesions respectively. This will be done as concisely as possible, the object being to present a brief summary of the distinctive features belonging to each of the

several classes of lesions. Lesions affecting the different valves or orifices will now be taken separately as points of departure, viz., mitral, aortic, tricuspid, and pulmonic lesions. These will be considered under distinct heads. The signs, symptoms, and effects of tricuspid and pulmonic lesions have thus far been passed by, owing to the comparative infrequency of their occurrence, exclusive of congenital malformations, and in order not to render the subject needlessly complicated to the student. It will suffice to present briefly the characters which belong to these lesions in this division of the subject. As involved in congenital malformations, they will be referred to hereafter.

Diagnostic Characters of Mitral Lesions.

Physical Signs.—An endocardial systolic murmur is present in the vast majority of cases, with the traits which distinguish a mitral regurgitant murmur, viz., its maximum of intensity at or near the apex of the heart, the intensity diminishing as the stethoscope is carried upwards over the body of the heart; generally feeble or lost above the base of the organ; not propagated into the carotids; often diffused over the left lateral surface of the chest, and not infrequently heard on the posterior surface, at the lower angle of the scapula, and in the interscapular space below the level of the spinous ridge of the scapula; the murmur more or less intense; generally soft, but sometimes rough.

The mitral portion of the valvular element of the first sound of the heart is often more or less impaired, or extinguished, the tricuspid portion of the same element remaining distinct, or abnormally intense. The aortic second sound is weakened; the pulmonic second sound is often intensified. Enlargement of the heart exists in the majority of the cases which come under observation. These signs characterize mitral valvular lesions involving insufficiency or regurgitation through the mitral orifice, *i. e.*, mitral regurgitant lesions.

A diastolic or pre-systolic murmur is present in a small proportion of cases; generally accompanied by a systolic mitral regurgitant murmur, but it may be present without the latter; its intensity greatest near the apex. Weakened aortic second sound and intensified pulmonic second sound are usually present, together with cardiac enlargement. These signs characterize, in general, contrac-

tion of the mitral orifice or mitral obstructive lesions. This variety of mitral lesions, however, is often unattended by a diastolic murmur; so that absence of this murmur is not evidence against the existence of the lesions.

The signs distinctive of mitral regurgitant and of mitral obstructive lesions are combined when these two varieties of mitral lesions coexist. Purring thrill is observed in a certain proportion of cases.

Symptoms and Pathological Effects.—Pain is rarely present. Abnormal force of the heart's action and palpitation denote consecutive enlargement, but these symptoms are often not prominent. The pulse is small and weak in proportion to the amount of obstruction or regurgitation; in an advanced stage, it becomes irregular and intermitting; irregularity of the pulse is, in some measure, characteristic of obstructive lesions. It is sometimes quick or vibratory. Turgescence of the jugular and other veins and dropsy occur at an advanced period when dilatation of the right cavities of the heart has been induced. Dyspnœa is more or less marked in proportion to the amount of regurgitation or obstruction, being more marked in cases of obstructive than regurgitant lesions. Cough and muco-serous expectoration occur frequently. Hæmoptysis is of frequent occurrence, and extravasation of blood in the lungs, or pulmonary apoplexy, takes place occasionally. Œdema of the lungs is a common event. All the symptoms and effects referable to the respiratory system are more marked when the lesions are obstructive than when they are only regurgitant.

In certain cases, lesions involving considerable and even great regurgitation or obstruction are remarkably latent and obscure as regards the symptoms and pathological effects. The diagnosis in these, as, in fact, in all cases, must rest mainly on the physical signs. On the other hand, lesions may exist, the existence and seat of which are determinable by physical signs, without involving much regurgitation or obstruction, and, consequently, not giving rise to symptoms or pathological effects. These lesions, so far as immediate danger is concerned, may be considered as innocuous.

Diagnostic Characters of Aortic Lesions.

Physical Signs.—An endocardial systolic murmur is present in the vast majority of cases, with the traits which distinguish an

aortic direct murmur; viz., its maximum of intensity at the base of the heart; the intensity diminishing as the stethoscope is carried downward over the body of the heart; comparatively feeble and often lost at the apex; propagated upward in the direction of the aorta, and often into the carotids; not diffused over the left lateral surface of the chest; and if heard on the posterior surface, either limited to, or most intense in, the interscapular space on and above the level of the spinous ridge of the scapula. The murmur more or less intense; generally soft, but sometimes rough, and occasionally musical. This murmur, when soft, is to be discriminated from inorganic aortic murmurs.

The aortic second sound of the heart is often weakened and indistinct; the pulmonic second sound is much less frequently intensified than in cases of mitral lesions. The mitral and tricuspid portions of the valvular element of the first sound retain their normal intensity, provided the lesions are limited to the aortic orifice. Enlargement of the heart exists in the majority of cases which come under observation. These signs characterize lesions with obstruction at the aortic orifice, *i. e.*, obstructive aortic lesions.

A diastolic murmur is present in a small proportion of cases, but in a larger ratio than in cases of mitral lesions; it is generally accompanied by a systolic aortic direct murmur, but it may be present without the latter; its intensity is greatest near the left margin of the sternum, on or about the level of the fourth rib. The aortic second sound is impaired in proportion as the valve is injured. The pulmonic second sound is less frequently intensified than in cases of mitral lesions. Cardiac enlargement is usually present. These signs characterize insufficiency of the aortic valve, or aortic regurgitant lesions.

The signs distinctive of aortic obstructive and aortic regurgitant lesions are combined when these two varieties of aortic lesions coexist. Purring thrill is observed more infrequently than in cases of mitral lesions.

Symptoms and Pathological Effects.—Pain is oftener present than in cases of mitral lesions, but is often absent. Abnormal force of the heart's action and palpitation, as a rule, are more prominent symptoms than in cases of mitral lesions. The pulse, in cases of considerable obstruction, is not notably reduced in size and strength; it is rarely irregular or intermitting, and still more rarely unequal. In cases of regurgitation it is quick, jerking, collapsing, and a longer

interval than natural is sometimes observed between the apex-beat or systolic sound and the pulsation in remote arteries. Visible pulsation of superficial arteries is frequently marked. Turgescence of the jugular and other veins, and dropsy, occur at a later period than in cases of mitral lesions, and are oftener wanting. Dyspnœa is less marked than in obstructive or regurgitant lesions of equal amount affecting the mitral valve and orifice. Cough and mucoserous expectoration and hæmoptysis are comparatively infrequent. Pulmonary apoplexy very rarely, if ever, occurs as a pathological effect. Œdema of the lungs is less frequent. All the symptoms and effects, in fact, referable to the respiratory system, are less frequent and marked than in cases of mitral regurgitant, and still less than in mitral obstructive lesions.

Lesions affecting the aortic as well as the mitral valve or orifice, and involving considerable obstruction or regurgitation, are in certain cases remarkably latent and obscure as regards the symptoms and pathological effects. The diagnosis rests mainly on the physical signs. Aortic lesions, also, may exist, and give rise to physical signs, without involving much or any obstruction or regurgitation, and are therefore innocuous as regards immediate danger.

The obstructive and regurgitant varieties of mitral and aortic lesions are found in various combinations in different cases. The diagnosis is then based on the union of the characters distinctive of the varieties severally. The physical signs characteristic of each variety can generally be distinguished in these combinations.

Diagnostic Characters of Tricuspid Lesions.

Physical Signs.—A systolic regurgitant murmur referable to the tricuspid orifice is rare even among the cases in which regurgitation through this orifice takes place. Regurgitation in consequence of widening of the tricuspid orifice, without a corresponding increase of the size of the valve, is not an uncommon result of enlargement of the right side of the heart consequent on mitral obstruction or regurgitation. The regurgitant current, however, rarely gives rise to a murmur, probably because the muscular power of the right ventricle is not sufficient to propel the current with force enough to produce audible vibrations. For the same reason a murmur is not always present in the exceedingly few instances of tricuspid regurgitation occurring in consequence of organic changes analo-

gous to those which affect the mitral valve. The rule, then, which is applicable to mitral lesions, viz., that a murmur is present in the vast majority of cases, cannot be applied to tricuspid lesions; and, hence, absence of murmur is not proof that the latter do not exist. A tricuspid regurgitant murmur, however, is sometimes observed. It is rarely, if ever, intense or rough, and is usually low in pitch. Its maximum of intensity is said to be at or above the xiphoid cartilage. It is heard within circumscribed limits, and is feeble or lost over the apex of the heart. In two instances of tricuspid lesions not associated with lesions affecting the left side of the heart, which have come under my observation, a soft and feeble systolic murmur was limited to the superficial cardiac region. Since tricuspid lesions, not congenital, are in most instances associated with lesions of one or more of the valves of the left side of the heart, a tricuspid regurgitant murmur, when present, accompanies a murmur, or murmurs, referable to the mitral or aortic orifice, or to both these orifices. It is to be distinguished from the latter by difference in pitch and quality, in addition to the difference of situation at which its maximum of intensity is observed.

Tricuspid regurgitation must diminish the intensity of the pulmonic second sound. If the tricuspid valve be injured, the tricuspid portion of the valvular element of the first sound must also be impaired.

A diastolic murmur may originate at the tricuspid orifice. Examples, however, of this murmur are among the rarest of the rare curiosities of medical experience, not only because contraction of this orifice is exceedingly infrequent, but also because the muscular power of the right auricle is insufficient to give rise to murmur in most of the instances in which the orifice is contracted. This murmur, theoretically, should be expected to be best heard at or just above the xiphoid cartilage.

Tricuspid obstructive lesions must diminish the intensity of the pulmonic second sound of the heart.

The combination of a systolic tricuspid regurgitant and a diastolic tricuspid direct murmur is possible. Dr. Walshe gives an example, based, however, on clinical evidence without the confirmation of a post-mortem examination.[1]

Free regurgitation through the tricuspid orifice, with great dilatation of the right auricle and hypertrophy of the right ventricle,

[1] Diseases of the Lungs and Heart, 2d London edition, p. 694.

may occasion a strong impulse felt at the base of the heart, to the right of the sternum, simulating aneurism. An example contained in the late work by Dr. Stokes is referred to in Chapter I.[1]

Symptoms and Pathological Effects.—Regurgitant and obstructive lesions, situated at the tricuspid orifice, do not produce those immediate effects on the respiratory system and the pulse which pertain to analogous lesions seated at the mitral orifice. They do not tend directly to give rise to dyspnœa, hæmoptysis, extravasation, etc., which are dependent on pulmonary congestion. They do not occasion irregularity, inequality, weakness, etc., of the pulse. Their immediate effects are manifested in the systemic venous system. Congestion of the systemic veins is a direct result proportionate to the degree of obstruction or regurgitation. Symptoms denoting this result are turgescence of the jugular and other veins; undulation and venous pulsation produced by the contraction of the right ventricle, and, in some instances, by the auricular contraction; lividity due to accumulation in the venous radicles. A pathological effect of the congestion of the systemic veins is general dropsy. This effect occurs more directly and at a much earlier period when tricuspid lesions exist, than when it depends on dilatation of the right cavities consequent on valvular lesions situated at the left side of the heart. Cerebral apoplexy is more likely to be dependent on tricuspid than on mitral or aortic lesions, exclusive of the instances in which this affection proceeds from fibrinous plugs detached from within the heart-cavities.

Diagnostic Characters of Pulmonic Lesions.

Physical Signs.—Lesions situated at the pulmonic orifice may give rise to a murmur with the first sound of the heart, which, following the plan pursued in naming the mitral and aortic murmurs, should be called a pulmonic direct murmur. This murmur has its maximum of intensity in the second or third intercostal spaces on the left side of the sternum, the situation where the pulmonic second sound of the heart is isolated from the aortic second sound. It may be propagated thence for a certain distance in a direction towards the left clavicle, but not in the direction of the aorta, and not heard over the carotids. To be considered as evidence of

[1] *Vide* page 54.

pulmonic lesions, not only must the murmur be referable to the pulmonic orifice, but it must be evidently an organic murmur. Attention to the several points already considered, will enable the auscultator to determine that it is not inorganic. It has already been stated that pressure over the pulmonary artery in young subjects, with the stethoscope, will sometimes develop a murmur in this vessel. Pressure from some cause within the chest may also cause a murmur referable to this artery. It has been observed in cases in which the pressure on the vessel was produced by an aneurismal tumor, a morbid deposit within the pericardium, enlarged bronchial glands, and a solidified portion of lung.[1] It must be difficult, in some instances, to eliminate these several sources of fallacy. A pulmonic murmur may be quite intense. I have met with an example of a musical murmur, systolic and diastolic, persisting through the whole beat, in other words, continuous, distinctly referable to the pulmonic artery, and so loud as to be heard with the ear in close proximity to, but not in contact with the walls of the chest.[2]

A diastolic murmur may accompany insufficiency of the pulmonic valve, constituting a pulmonic regurgitant murmur. It must be difficult to distinguish between this and an aortic regurgitant murmur, except it be accompanied by a pulmonic direct murmur, and not by an aortic direct murmur. In the vast majority of the cases in which a diastolic murmur is present, it is either an aortic regurgitant, or a mitral direct murmur.

Lesions involving injury to the pulmonic valve must impair the intensity and distinctness of the pulmonic second sound of the heart.

A pulmonic direct and a pulmonic regurgitant murmur may be associated in the same case, or either may be present without the other. Pulmonic lesions, however, exclusive of congenital malformations, are so rare, that the opportunities of any clinical observer, however large his experience, for studying the physical signs are extremely limited. Hypertrophy of the right ventricle, which is produced by obstructive or regurgitant lesions of the pulmonic orifice, involves augmented intensity of the tricuspid valvular element of the first sound, and an impulse in the epigastrium.

[1] Bellingham, op. cit., part ii. p. 386. Da Costa in Am. Journ. of Med. Sciences, January, 1859.

[2] Case of Kelly, Private Records, vol. x. p. 577.

Symptoms and Pathological Effects.—The primary effect of obstructive or regurgitant lesions situated at the pulmonic orifice is enlargement of the right ventricle. The secondary and remote effects, and the symptoms thereon dependent, are essentially those which are occasioned by tricuspid lesions, being due to distension of the right auricle, tricuspid regurgitation, and congestion of the systemic veins.

Treatment of Lesions affecting the Valves and Orifices of the Heart.

With reference to the management of patients affected with chronic valvular lesions, several important considerations, which have been already presented, are to be kept in mind.

1. The anatomical changes which the valves and orifices have undergone are irremediable, and therefore do not claim any special medicinal treatment. The existing lesions must remain. The damage which they have occasioned cannot be repaired. Medication employed for that object will be worse than useless. The morbid processes giving rise to the lesions, have occurred long before the symptoms of an organic affection of the heart became developed. In the majority of cases the origin of the affection may be dated at an attack of acute rheumatism several years prior to the period when ailments referable to the heart are first experienced. The changes incidental to these processes have, in the mean time, been slowly progressive. They will, in all probability, continue to progress, involving more and more damage. This we cannot expect to prevent, but something can be done to retard their progress, and, more especially, to control their primary effects.

These facts not being always sufficiently appreciated, practitioners sometimes employ mercury and other remedies called alteratives, with a view to the removal of morbid material deposited on or about the valves. I have met with cases in which depletion, low diet, counter-irritation, etc., were resorted to, under the idea that the lesions involved persisting chronic inflammation of the endocardial membrane. These measures can hardly fail to aggravate the cardiac symptoms, and to expedite effects which it is a great object of the management to postpone as long as possible.

2. Lesions may exist, giving rise to murmurs more or less intense, without producing any immediate morbid effects, not involv-

ing either obstruction or regurgitation. Such lesions may remain for an indefinite time innocuous, but there is a liability of the changes incidental to them leading, at some future period, to serious results. These cases, therefore, claim a certain amount of watchfulness and supervision. The existence of a cardiac murmur dependent on innocuous lesions is often ascertained by accident, there being no symptoms of disease referable to the heart. I have repeatedly met with it in examining persons who considered themselves in perfect health. These persons are in no immediate danger, and it would give rise to needless alarm to inform them that they have an organic affection of the heart, since it is a common notion that any such affection involves liability to sudden death. There is, however a prospective danger not to be overlooked. It has occurred to me in two instances to examine for life insurance persons presenting an organic murmur, without other evidence of cardiac or other disease, who were, of course, not deemed proper subjects for insurance; but they succeeded in obtaining policies in other companies, and both have since died with well-marked disease of the heart.

3. Even when lesions exist which do involve more or less obstruction or regurgitation, it does by no means follow that the immediate danger is great. This statement holds good in some cases in which there is, at the same time, considerable enlargement of the heart. I am acquainted with several persons who do not consider themselves as invalids, some being engaged in active business, in whom the existence of organic murmur, with cardiac enlargement, was ascertained many years ago.[1] In such cases the organic affection does not call for active therapeutical measures, but knowledge of the existence of the cardiac affection is highly important to the practitioner, and should influence his advice as regards habits, regimen, etc., as well as his treatment of intercurrent diseases. The tolerance of lesions in some instances is truly remarkable. A boy, aged eleven years, who recently came under my observation, presenting three organic murmurs, viz., a mitral regurgitant, an aortic direct, and an aortic regurgitant, with much cardiac enlargement, the præcordia projecting and the apex beating half an inch without the nipple, not only made no complaint of symptoms referable to the heart, but was able to take violent exer-

[1] *Vide* Essay on Clinical Study of Heart-Sounds, *Trans. Am. Med. Association*, vol. xi.

cise, and to engage in rough sports with apparently as much ease as any of his companions. In this as in other instances in which lesions involving more or less obstruction or regurgitation are borne without notable inconvenience, the physical signs denoted enlargement by hypertrophy, not by dilatation, and the action of the heart was vigorous.[1]

4. The enlargement of the heart being, as a rule, proportionate to the amount of obstruction or regurgitation, or both, occasioned by valvular lesions, the abnormal size of the heart may, in general, be taken as a criterion of the importance to be attached to the lesions. So long as the heart is not much enlarged, the patient is exposed to only certain contingent evils incident to the lesions—for example, arterial obstruction from detached deposits. Exclusive of accidental events and associated affections, the symptoms and remote effects of obstructive and regurgitant lesions correspond to the primary effects of these lesions, as denoted by the increased bulk of the heart. This statement holds good in the majority of cases, but, as already stated, the rule is not without exceptions.

5. The secondary and remote effects of valvular lesions, as a rule, are not developed so long as the enlargement of the heart is by hypertrophy, unless, from some cause, weakness of the organ has been induced. Obstructive and regurgitant lesions tend first to produce hypertrophy. The muscular walls increase in thickness up to a certain limit. When this limit is reached, dilatation of the cavities ensues, and, finally, predominates over the hypertrophy. The increased power of the organ, incident to the hypertrophy, compensates for the immediate consequences of obstruction and regurgitation. The hypertrophy is, in fact, a conservative provision to obviate the evils of obstructive and regurgitant lesions. The patient is comparatively safe while hypertrophy predominates. The secondary and remote effects are incident to the dilatation which takes place after the hypertrophy has reached its limit. The immediate danger, other things being equal, is proportionate to the amount of predominance of the dilatation. This is because the heart is weakened in proportion to the predominance of dilatation. Weakness of the organ, due to other causes than dilatation, will also favor the development of the secondary and remote effects of valvular lesions. These facts are of great importance in their bearing on the treatment of patients affected with these lesions.

[1] Case of Horan, New Orleans Charity Hospital. H. Reports, vol. xiii. p. 84.

In view of the foregoing considerations, the main objects of treatment which relate directly to the condition of the heart, in the early stage of valvular lesions, are, 1st. To prevent, or, as far as possible, to retard the progressive anatomical changes incident to the existing lesions; and, 2d. To obviate the tendency to weakness and dilatation of the heart.

The anatomical changes seated in the valves and orifices, give rise to the varied morbid appearances which have been mentioned[1] in Chapter third. These changes cannot be reached by any special remedies. Their progress can only be indirectly affected by preventing overstraining of the valves, which must occur whenever the organ is unduly excited or overtasked, and by avoiding the causes which favor renewal of inflammation of the endocardium. Excessive muscular exercise, great mental excitement, the intemperate use of alcoholic stimulants, etc., promote the progress of valvular lesions, by exciting unduly and overtasking the heart. Patients with valvular lesions devoid of immediate danger, should pursue a course of life which, as far as practicable, will be exempt from causes inducing great disturbance of the circulation. In pointing out the regimen, habits, etc., however, the importance of fostering the muscular power of the heart, to which reference will be presently made, is not to be lost sight of. The causes favoring the development of endocarditis, are, in general, those which tend to give rise to rheumatism, since in the vast majority of cases endocardial inflammation is of rheumatic origin. Unusual exposure to the vicissitudes of the weather are regarded, perhaps justly, as often determining an attack of rheumatism when the diathesis exists. The existence of the diathesis is shown by the previous occurrence of one or more rheumatic attacks in the majority of the persons affected with valvular lesions, the origin of the latter being referable, in such cases, to a former attack of rheumatism. A fresh attack exposing the patient to a renewal of the endocarditis, is a calamity to be averted, if possible, by avoiding the exciting causes so far as these are appreciable. In fulfilling this object of treatment, hygienic regulations are chiefly involved. Judicious management will undoubtedly do something toward rendering the progress of the lesions more slow than would otherwise be the case; but we can hardly expect to arrest their progress. If, however, they are very slowly progressive, life and comfortable health may be prolonged for an indefinite period, perhaps even to an advanced stage.

[1] *Vide* page 120.

The same hygienic regulations are equally important with reference to the second object, viz., to obviate the tendency to weakness and dilatation of the heart. The judicious management of patients affected with valvular lesions prior to the development of the secondary and remote effects of these lesions, depends in a great measure, on a proper appreciation of this object. It is commonly said by writers on diseases of the heart, that the treatment of valvular lesions virtually resolves itself into that designed to prevent and diminish enlargement of the heart. This involves an important error as well as an important truth. It is highly desirable to prevent dilatation but not to arrest hypertrophy. On the contrary, if enlargement must occur as a result of obstructive or regurgitant lesions, hypertrophy is to be encouraged, if by so doing, dilatation may be prevented. The serious evils of valvular lesions, as we have seen, occur when the limit of hypertrophy has been reached and dilatation predominates. In the predominance of hypertrophy may be said to consist, in a great measure, the security of the patient. This remark is also applicable to the muscular power of the heart. So long as the organ acts with vigor, the secondary and remote evils are deferred. Weakness of the heart leads to these evils. Weakness predisposes to dilatation, and, conversely, dilatation involves weakness. To prevent weakness and dilatation, then, in the early stage of valvular lesions, is the great object of treatment so far as it relates directly to the condition of the heart.

Undue excitement and overtasking of the heart induce weakness and favor dilatation. The muscular power here, as in other situations, is exhausted by too great exertion, and the walls yield more readily to distension, under these circumstances, from the accumulation of blood within the cavities. The causes, already referred to, which excite unduly and overtask the heart, viz., excessive muscular exercise, mental excitement, the intemperate use of alcoholic stimulants, etc., are, therefore, to be avoided with respect to the second, not less than the first object of treatment. Exercise, however, within certain limits, is highly important with a view to the preservation of the power of the heart's action. Patients affected with obstructive or regurgitant lesions will retain a compensatory vigor of the heart, and the epoch when dilatation succeeds hypertrophy will be postponed for a longer period by habits which involve a judicious amount of exercise than by a life of complete repose. Active occupations, whether pursued as a calling or for amusement, or with reference merely to exercise,

should not be abandoned. Persons under the necessity of performing daily manual labor will do better to continue to work, so far as they are able without inconvenience, than to become fixtures in the wards of a hospital. They who are above this necessity should either follow some active pursuits or engage in sports which demand a certain amount of physical activity. Indolence or inaction of the muscular system tends to produce weakness of the heart and favors fatty degeneration, thereby contributing to the production of dilatation rather than hypertrophy. The rules which should govern exercise have already been considered in connection with the treatment of hypertrophy, to which the reader is referred.[1] These rules are applicable to cases of valvular lesions, with or without hypertrophy. They are, of course, not to the same extent applicable to cases in which the lesions have already led to dilatation.

The diet suited to obviate a tendency to weakness and dilatation is that best adapted to healthy nutrition. Healthy nutrition, and thereby the muscular vigor of the heart, require blood rich in nutritive materials. A poor and insufficient diet tends to hasten the evils resulting from valvular lesions. The diet should embrace a fair proportion of animal food. Liquids should be taken sparingly, the object being to secure a good quality, but not to increase the quantity of blood. Restrictions, as respects fatty substances and those readily converted into fat, are important if there are grounds to suspect a disposition to fatty degeneration. The articles of food should be adapted to the digestive powers. The action of the heart, as is well known, is liable to be disturbed through its sympathetic connection with the stomach, when digestion is labored or imperfect. Dyspeptic disorders will claim appropriate treatment. Tonics and stimulants, in moderate quantity, are indicated whenever the digestive powers are enfeebled. Exercise in the open air, within proper limits, is important with reference to its influence on digestion. Cheerfulness and mental recreation are desirable for the same end.

Opposite conditions of the blood alike tend to weakness and dilatation, viz., plethora and anæmia. If the blood be too abundant and the red globules in excess, the heart is overtasked and unduly stimulated. Bloodletting, under these circumstances, may be appropriate. But it should be employed with discrimination

[1] Chap. I. page 72.

and great circumspection, inasmuch as the impoverishment caused by its injudicious employment is a condition worse than plethora. In general, other methods of depletion, which are not spoliative, are to be preferred, viz., saline laxatives and diuretics, in conjunction with a dry diet. Anæmia is a far more unfavorable condition than plethora, and claims efficient treatment with chalybeate tonics, nutritious diet, etc. The symptoms referable to the heart, in some cases of valvular lesions, are, in a great measure, due to functional disorder incident to anæmia, and when the anæmic condition is removed, all the symptoms may disappear. This fact should be borne in mind. The practitioner is liable to consider all the symptoms as resulting directly and exclusively from the lesions, and, consequently, is led to exaggerate the immediate danger from the latter. Patients who suffer much from palpitation, etc., when anæmia is conjoined with valvular lesions, may experience no inconvenience when the blood is restored to its normal condition. This is intelligible in view of the well-known fact that anæmia often gives rise to functional disorder of the heart when this organ is free from organic disease.

The treatment of valvular lesions, as thus far considered, has reference to the condition of the heart prior to the period when dilatation has ensued, either enlargement of the organ not having taken place, or hypertrophy being as yet predominant. The secondary and remote effects of valvular lesions, as has been seen, for the most part occur after dilatation predominates over hypertrophy. It remains to notice the treatment due to the condition of the heart at this stage, and the treatment of the secondary and remote effects.

So far as the heart is concerned, the treatment at this stage is essentially that which has been already considered in connection with the subject of dilatation.[1] Extrinsic circumstances affecting the circulation, such as exercise, mental emotions, etc., now occasion symptoms of disturbance much more marked, and attended with far greater inconvenience. The ability to take exercise without palpitation and dyspnœa is diminished, and quietude may be indispensable. Within the limits, however, to which exercise may be borne without discomfort, it is still desirable. A nutritious, sustaining diet is not less indicated. Attention to the condition of the stomach is equally important. Bloodletting is much more

[1] *Vide* Chap. I. page 85.

rarely, if indeed it be ever, called for. Plethora, if it exist, claims methods of depletion which are not spoliative. Anæmia demands the same efficient measures. The general object is to increase, if possible, the muscular power of the heart. It is doubtful whether this object is promoted by any remedies which exert a direct, special effect upon the heart. Nux vomica or strychnia, given in minute doses and long continued, is, however, advocated by Dr. Corson as a remedy having such an effect.[1]

At this stage, not only is the heart enfeebled, but the rhythm of its action is often disturbed, as denoted by irregularity, intermittency, and inequality of the pulse. Remedies designed to tranquillize and regulate the movements of the organ are now indicated. For this end, digitalis often proves a valuable remedy. Care is to be taken not to give this remedy to the extent of retarding too much the heart's action. Observing proper caution in this respect, the action of the heart not only becomes more regular under its use, but the contractions of the ventricles appear to take place with greater power and completeness, as denoted by increased fulness and force of the pulse. It is proper to add that this statement, as regards the value of digitalis, is in opposition to the views of some distinguished authors, who regard it as rarely useful and attended with hazard. Its usefulness and freedom from danger turn on the influence which it exerts on the power of the heart's action. The opinion which I have expressed is based on inferences drawn from clinical observations. Belladonna, the hydrocyanic acid, aconite, and sometimes opium in small doses, are other remedies which may be found useful in fulfilling this indication.

A large share of the secondary and remote effects of valvular lesions are dependent on congestion. The lungs are generally first and most affected; afterwards, the brain and abdominal viscera. The tendency to congestion of internal organs is obviated most effectively by measures which prevent weakness and dilatation of the heart, or which increase its muscular power if these primary effects have already taken place. In addition, something may be effected by promoting, as far as possible, the circulation in the extremities and at the surface of the body, and by revulsive measures. The body should be protected by sufficiently warm clothing, and prolonged exposure to cold should be avoided. Friction of the surface and stimulating pediluvia are useful in fulfilling this indi-

[1] New York Journal of Medicine, May, 1855.

cation. Rubefacient applications and dry cupping are the appropriate revulsives.

Dyspnœa, cough, and expectoration, often claim special attention. The suffering from a sense of breathlessness, frequently severe, must be palliated by the remedies known as antispasmodics, particularly the ethereal preparations, and by anodynes, in addition to revulsive measures. Palliation, in most instances, is all that can be expected from treatment. Cough, exceeding that required for expectoration, may be allayed by such remedies as hyoscyamus, conium, hydrocyanic acid, or by small doses of opium. Muco-serous expectoration is sometimes a mode of relief, being a sort of local, spontaneous depletion, and is to be encouraged rather than arrested. Superinduced or intercurrent pulmonary affections, such as bronchitis, pneumonia, and pleurisy, demand appropriate treatment, but bloodletting and other measures which tend to weaken the heart, are to be employed with great circumspection. The coexistence of valvular lesions and dilatation generally renders sustaining measures more than ever important in the management of these affections.

The importance of correcting disorders of digestion, and improving this function when impaired, is not less in the advanced than in the early stage of valvular lesions. Mercury is often prescribed with a view to relieving congestion of the liver by increasing the secretion of bile. Granting that it has this effect, it is a remedy of doubtful propriety if given so as to incur risk of mercurialization. As an occasional laxative, or cathartic, it is admissible. Constipation is to be avoided, and moderate purgation, from time to time, affords relief as a means of local depletion when the digestive organs are suffering from congestion. Purgatives too often repeated, however, will do harm by depressing the vital powers, and thereby weakening the heart.

For the relief of cerebral congestion, which we have seen occasions inconvenience and evils less frequently than is generally supposed, reliance must be had on the revulsive measures, in addition to those which relate directly to the condition of the heart.

General dropsy is a remote effect occurring in a large proportion of the cases of valvular lesions which are prolonged to an advanced stage. It is usually evidence of a degree of weakness and dilatation, precluding expectation of permanent improvement, and denoting that a fatal termination is not far distant. But in some instances complete relief is obtained, and the dropsy may not recur for a

considerable length of time. These are instances in which the dropsy has been promoted by associated morbid conditions, such as an anæmic state of the blood, or by extrinsic causes which have temporarily enfeebled the heart. The event is not only important as a symptom, but it imposes inconvenience and suffering proper to itself, and hastens a fatal issue. It calls, therefore, for appropriate treatment.

The immediate objects of the treatment of cardiac dropsy are the resorption of the effused liquid, and, at the same time, increased power and completeness of the ventricular contractions. Resorption is to be effected, if possible, by eliminating water from the blood by means of diuretics or hydragogue cathartics, conjoined with a dry diet. In the selection of diuretics, those are to be preferred which increase the quantity of urine without increasing, proportionably, its solid constituents; in other words, those which eliminate especially water. Experimental observations render it probable that different diuretics differ in this respect, digitalis, juniper and squill, for example, increasing the flow of urine, while the amount of solid matter is below that of health.[1] It is as important in the treatment of dropsy by diuretics not to eliminate solids, as the latter is the object of treatment with a view to depuration in various affections. In the employment of diuretics and hydragogue cathartics, more especially the latter, great care is required not to push the remedies to an extent to lower too much the powers of the system, and thereby weaken the action of the heart. While measures are pursued to effect resorption, the second object of treatment should not be lost sight of, viz: to increase the power and completeness of the ventricular contractions. This object involves nutritious diet, tonics and exercise within proper limits.

The choice between diuretics and hydragogue cathartics will depend on the readiness and extent to which the kidneys respond to the former of these two classes of remedies. Different cases differ much in this respect. In some instances hypersecretion of urine is easily effected; in other instances with difficulty, and not to the extent desired. Reliance must then be had on cathartics. In general, diuretics should be first tried, and they are to be preferred if

[1] On the Action of Certain Vegetable Diuretics. By William A. Hammond, M. D., assistant surgeon U. S. Army, *Am. Journ. of Med. Sciences*, No. for Jan., 1859. This is an interesting and important subject for further experimental observations.

found to operate satisfactorily. The general principle involved in the selection of diuretics has been stated, but we have not, as yet, sufficient facts to establish a division of all the numerous articles which induce diuresis into those which do and those which do not increase the solid constituents of the urine. As regards the diuretic effect, we have to be guided, in a great measure, by experimental trials in individual cases. A diuretic remedy may act efficiently in one case and prove inefficient in another case, a different article being found to act satisfactorily in the latter. All practical physicians must have been led to notice this fact. Usually, different diuretics act better in combination than separately. Digitalis and squill, for example, may be given in combination, and, at the same time, the bitartrate, the nitrate, or the acetate of potash, largely diluted in an infusion of juniper, parsley, or fleabane. As remarked by Prof. Wood, diuretics may fail at a particular period, and act efficiently at another period in the same case. The ingestion of liquids should be restricted so far as due regard to comfort will permit. The mode by which diuretics lead to resorption being the elimination of water from the blood, which involves an increase of the density of the latter, it is plain that their efficacy in relieving dropsy will be limited or rendered nugatory by the free introduction of liquid into the system.

Prof. Christison has advocated the external use of diuretics in certain cases. I have repeatedly tried this method, and generally without much success. In one instance, however, which came under my observation, the patient being under the care of my friend, Prof. J. P. White, the effect was remarkable. Diuretics given internally having lost their effect in this case, a liniment composed of equal parts of the tinctures of squill, digitalis, and iodine, and two parts of soap liniment, was applied freely, with considerable friction, over the abdomen and thighs. The patient took no remedy in addition except the iodide of iron. Under this treatment, in a week, he lost nine pounds in weight, the secretion of urine being greatly increased. The anasarca disappeared, and did not again return for several months, the comfort and general health of the patient becoming in the mean time much improved.

Hydragogue cathartics are to be employed when diuretics fail to act satisfactorily or to accomplish the end desired. Of the different articles embraced in this variety of cathartic remedies, elaterium is the most efficient. The great activity of this article, when pure, requires care in its administration. From a sixth to a quarter of a

grain may be directed every one or two hours till a sufficient number of watery stools are procured. The intervals between the days of its administration must be regulated by the state of the patient and the prostration occasioned by its operation. The bitartrate of potash, given in pretty large doses in a concentrated solution, frequently acts efficiently. This remedy and jalap form an efficient combination.

The treatment of cardiac dropsy is sometimes remarkably successful. The effused liquid is rapidly and completely absorbed. The patient experiences so much relief that he is encouraged to hope for recovery. This the physician does not expect, but he may hope to postpone the recurrence of dropsy by strengthening the heart and removing causes which exist in addition to the obstruction due to the cardiac affection. Anæmia predisposes powerfully to dropsical effusion. The restoration of the blood to its normal condition may secure long exemption from recurrence of the dropsy. If, however, the dropsy be altogether dependent on the obstruction caused by the cardiac affection, some degree of palliation is all that is to be hoped for. The accumulation of effused liquid remains and augments, often, notwithstanding appropriate measures of treatment. This is more likely to be the case if the patient be obliged to remain much of the time in the sitting posture on account of dyspnœa. The coexistence of Bright's disease also lessens greatly the prospect of relief, but this combination, as has been seen, is less frequent than is generally supposed. Temporary relief is sometimes obtained by puncturing the lower extremities, water draining away in abundance through the artificial openings. Numerous minute punctures may be made with a fine needle, not deep or large enough to occasion either pain or hemorrhage. These may be frequently repeated. I have not observed unpleasant results from this mode of making punctures. If the size of the punctures be sufficient to cause visible wounds, there is a liability to erysipelatous inflammation and gangrene, which renders the operation of doubtful expediency. This remark is also applicable to incisions, which some writers have advised. The great distension of the integument of the lower extremities in some instances gives rise to fissures and ulcerations, through which the effused liquid freely escapes. When these occur, it is not wise to attempt to heal them promptly.

An important part of the management of cases of valvular lesions relates to the communications on the subject to be made to the pa-

tient. An endocardial organic murmur which is discovered incidentally in the examination of a patient, need not be announced, for, if the heart be not enlarged, the danger is prospective, perhaps remote, and gratuitous uneasiness may be occasioned by the patient being made acquainted with the existence of an organic affection. Unsoundness of the heart is generally supposed to be, in all cases, a very serious matter, and to involve liability, at any moment, to sudden death. Some practitioners, participating in this popular impression, injudiciously apprise patients that they must expect to be taken off without warning. I have met repeatedly with instances in which persons have been so informed, much to the prejudice of their comfort, usefulness, and even their prospect of preserving comfortable health for a long period. It should be borne in mind that lesions which give rise to murmurs are often innocuous, the danger being prospective, and perhaps remote. And even when the lesions are of a nature to involve obstruction or regurgitation, and have led to considerable enlargement of the heart, life and comfortable health may be preserved for many years. Moreover, statistics show that sudden death occurs in only a small proportion of the cases of organic disease of the heart, often involving, when it does occur, some associated morbid condition—for instance, structural degeneration of the cerebral arteries, leading to rupture and extravasation. Cases of fatal apoplexy occurring in persons with cardiac lesions are frequently incorrectly explained by imputing the occurrence wholly to the condition of the heart.

If the attention of the physician be called by the patient to the state of the heart, and an opinion requested, the existence of lesions cannot be denied. Truth, and justice to the physician himself, as well as good faith toward the patient, require that the fact should be candidly stated. The statement then should be accompanied by such explanations as will serve to divest the fact of greater importance than really belongs to it. If proper pains be taken, this, happily, is not difficult, since the mental condition incident to disease of the heart generally leads patients to accept the most favorable view of the case which can be conscientiously submitted.

The prognosis, to the friends of the patient, should be cautiously given. The duration of life, except in cases of advanced disease, is extremely variable. There is, on the one hand, a liability to certain accidents and incidental affections which may prove fatal unexpectedly; and, on the other hand, patients often live for a long time after the signs and symptoms denote lesions of a most serious character.

CHAPTER V.

CONGENITAL MISPLACEMENTS, DEFECTS, AND MALFORMATIONS OF THE HEART.

Transpositions and expositions—Ectopia pectoralis cordis—Ectopia cordis ventralis—Ectopia cordis cephalica—Deficiency of the pericardium—Biloculate heart—Heart with three cavities—Deficiency of auricular and ventricular septa—Obliteration of, and obstruction at, the pulmonic artery—Supernumerary septum—Patency of the foramen ovale and the ductus arteriosus—Deficiency and excess of segments of semilunar valves—Union of curtains of the mitral and tricuspid valves—Diagnosis of malformations—Causes of death—Coexistence of tuberculosis—Treatment.

Cyanosis. Definition—Its connection with different malformations—Conclusions respecting the mode of its production—Cyanotic phenomena in various affections exclusive of malformation of the heart—Diversities as respects degree and extent of the cyanotic discoloration—Other variations—Associated symptoms—Diagnosis—Prognosis—Treatment.

The various malformations, etc., of the heart are especially interesting in their relations to the physiology of the circulation and to embryology. A general knowledge of this subject, however, is important to the physician. It is desirable for the practitioner to recognize the existence of congenital affections, by means of signs and symptoms, during life, and, as far as practicable, to discriminate between them. The study of these affections has an important bearing on a subject which will, in consequence, be considered in this chapter, viz., cyanosis.

Treating of the different varieties of congenital affections only so far as is consistent with the practical scope of this work, they will occupy but a small space. I shall follow the philosophical arrangement adopted in the late treatise by Dr. Peacock, to which the reader desirous of a fuller consideration of the subject is referred.[1] The facts relating to this subject which will be presented are derived mainly from the author just named.

[1] On Malformations, etc., of the Human Heart. With Original Cases. By Thomas B. Peacock, M. D., Fellow of the Royal College of Physicians, etc. London: John Churchill, 1858.

CONGENITAL MISPLACEMENTS OF THE HEART.

The misplacements are either of *transposition* or *exposition*. Instances in which the heart is situated in the right side of the chest, are not so infrequent but that numerous examples have been reported. In some instances the other viscera are likewise transposed, and in other cases the situation of the heart alone is abnormal. This abnormity is not incompatible with health and long life. Removal of the heart into the right side, as is well known, occurs as a result of pleurisy, with a large amount of effusion, affecting the left side. The organ may form attachments and remain fixed in the right side after the liquid effusion has been absorbed. A similar result sometimes follows absorption of a large amount of liquid effusion into the right pleural sac. The existence of pleuritic effusion is readily ascertained by means of the physical signs, and the permanent changes in the size and configuration of the affected side enable the diagnostician to determine the fact of its previous existence. In either case the misplacement of the heart is not to be considered as congenital.

In cases of *exposition*, the heart may be situated exterior to the chest. These instances constitute the variety known as *ectopia pectoralis cordis*. In none of the instances cited by Dr. Peacock in which the heart alone was exterior to the chest, did life continue over forty hours after birth. This variety of ectopia is extremely rare, excluding the cases in which, at the same time, the viscera of the abdomen are protruded. They offer valuable opportunities for studying the movements of the heart by means of the sight and touch. In another variety of exposition, the heart is situated below the diaphragm, and in these cases there may or may not be an external tumor. This variety, called *ectopia cordis ventralis*, is not incompatible with long life and vigorous health. In another curious variety, the heart is situated in the front of the neck. This is called *ectopia cordis cephalica*. In all the cases reported of this variety, the infants have died shortly after birth. These different misplacements may be readily ascertained by inspection, palpation and auscultation. But in cases of *ectopia cordis ventralis*, the malposition may occasion so little inconvenience that attention may

not have been directed to it during life, and the anomaly, therefore, is not ascertained before death.

DEFICIENCY OF THE PERICARDIUM.

In some of the cases reported by the older anatomists as examples of congenital absence of the pericardium, it is probable that there existed pericardial adhesions, and the deficiency was only apparent, not real. But the pericardium is undoubtedly sometimes wanting. Dr. Baillie described an instance which came under his observation incidentally, the chest having been opened to explain to a class of students the normal situation of the thoracic viscera. The heart was bare, lying loose in the left cavity of the pleura. In this instance there had been no morbid symptoms referable to the heart during life. In several other examples cited by Dr. Peacock, no troubles pertaining to the heart or circulation had existed. In one instance the patient died at the age of seventy-five years with disease of the aortic valve. There are no diagnostic characters by which the existence of this abnormity can be determined during life.

MALFORMATIONS OF THE HEART.

The malformations of the heart are dependent, for the most part, on arrest of development at different periods of fœtal life. Examples of the biloculate heart, *i. e.*, the heart consisting of a single auricle and ventricle, are rare, but a considerable number of authentic cases have been reported by different observers. This abnormity is compatible with only a short duration of life, death occurring, in general, a few hours or days after birth. The pulmonic vessels in these cases are given off by the aorta, the venæ cavæ and pulmonic veins terminating in the single auricle. In this variety of malformation, the arrest of development takes place early in fœtal life. Cases in which the heart consists of only three chambers are not so rare as the preceding. They denote an arrest of development occurring at a later period of intra-uterine exist-

ence. Persons with this malformation have lived for several years, but generally death takes place within a few weeks or months. The cavities are two auricles and a single ventricle, the latter presenting sometimes a rudimentary septum. These cases differ as respects the arrangement of the primary vessels. In some cases, both the aorta and the pulmonic artery spring from the single ventricle; in others, the aorta gives origin to the pulmonary vessels, or, if the pulmonic artery exist, it is in a rudimentary form, and the blood is supplied to the lungs through the ductus arteriosus. Deficiency of the ventricular and auricular septa, either or both, is a variety of malformation vastly more common than the two varieties just noticed. When the ventricular septum is more or less deficient, the imperfection generally, but not invariably, exists at the base. The explanation of this is, the division of the cavities is here effected last during fœtal life. Hence, this form of malformation indicates an arrest of development occurring at a period still later than in the two previous forms. In the fully developed organ there exists at the upper part of the septum a triangular space in which the ventricular chambers are only separated by the endocardium and fibrous tissue on the left side, together with the lining membrane and a thin layer of muscular tissue on the right side. The average length of the sides of this triangle is about seven lines, and the base is somewhat wider. This is sometimes distinguished as the *undefended space*. It has been a question with pathologists whether, in a certain proportion of the instances of deficiency at this portion of the interventricular septum, it be not due to rupture or perforation after birth. Bouillaud contends that it is frequently attributable to disease. Dr. Peacock concurs in the opinion that it is thus attributable in some cases, but he thinks that the proportion is smaller than that claimed by the French pathologist just named. Deficiencies in both the ventricular and auricular septa are usually associated with other defects, and especially with obstruction at the pulmonic orifice. The former are probably due, in a great measure, to the latter. The effect of pulmonic obstruction on the blood-currents prevents that complete separation of the cavities (exclusive of the foramen ovale) which should take place during the latter part of fœtal life. Considerable deficiency of the partitions between the ventricles or auricles is not uniformly attended by marked symptoms referable to the heart. Persons may present few or no indications of the existence of any heart affection. If the deficiency

in the septa be associated with mal-arrangement of the primary vessels, the consequences are far more serious. The pulmonic artery, as well as the aorta, may spring from the left ventricle. Virtually, in such cases, the heart consists of three cavities. The left ventricle becomes greatly enlarged, and the right ventricle proportionately atrophied. Life has continued for years under these circumstances. In other cases, the aorta, as well as the pulmonic artery, arises from the right ventricle. The pulmonic orifice in these cases is usually obstructed; the foramen ovale remains open, and the ductus arteriosus occasionally continues pervious.

Obliteration of the pulmonic artery would at first seem to be an abnormity incompatible with life. Several cases, however, have been reported in which death did not occur for several years. In connection with this malformation, the interventricular septum is often defective. The ductus arteriosus generally remains pervious, and the pulmonary vessels are supplied through this channel. If the ventricular septum be complete, the foramen ovale continues open. The open foramen and the defect in the septum between the ventricles, instead of adding to the danger, afford relief to the overloaded right ventricle and auricle, without which life would probably not continue, except for a brief period. The right ventricle becomes greatly dilated and hypertrophied in this variety of malformation.

The presence of a supernumerary septum in the right ventricle constitutes another variety of malformation. This superfluous septum may be so far complete that the heart appears to have three ventricles. In its effects, this malformation is essentially similar to those involving obstruction or obliteration of the pulmonic orifice, and with the latter malformations it is often associated. The foramen ovale and ductus arteriosus, either or both, are generally open, and the septum between the ventricles is, in some instances, deficient. The duration of life varies according to the amount of obstruction. In the cases collected by Dr. Peacock, death occurred between the ages of nine and thirty-six years.

Certain malformations consist in the non-occurrence of those changes which should ensue after birth. The most important of these are patency of the foramen ovale and of the ductus arteriosus. These passages, peculiar and essential to the circulation in fœtal life, remain patent, one or both, after birth, in most of the instances in which the ventricular septum is more or less deficient, and, in general, their patency is associated with obstruction at or near the

pulmonic orifice. When the latter condition coexists, the right ventricle generally becomes hypertrophied, but to this rule there are exceptions. If the communications between the auricles and the primary arteries are free, the right ventricle, instead of being enlarged, is sometimes found to be quite small, evidently atrophied, the blood finding a ready outlet through the fœtal passages, thus preventing accumulation within the right ventricular cavity. It is to be remarked that although in the great majority of instances of open foramen ovale, obstruction at or near the pulmonic artery is associated, the rule is not invariable. Hence, this obstruction, although a frequent, is not the sole cause of the persistence after birth of the communication between the auricles. Patency of the ductus arteriosus is also a conservative provision in cases of obliteration of, or great obstruction at the pulmonic orifice. It is equally so in certain instances in which the foramen ovale becomes closed during fœtal life. This duct remains pervious in some cases in consequence of obstruction at the aortic, and also at the mitral orifice. These coexisting malformations account for the persistence of the open duct in the great majority of cases, but the latter is sometimes observed when it is not thus to be explained, existing independently of other abnormities to which a relation of dependency can be traced.

Finally, there are certain malformations which do not interfere with the functions of the heart, but which may lay the foundations of disease in after life. Under this head are embraced, on the one hand, deficiency, and, on the other hand, excess in the number of segments of the semilunar valves. Deficiency is of more importance than excess. In fact, it does not appear that the latter leads to any serious consequences. The former involves, in certain cases, insufficiency and regurgitation, and, probably, a disposition to take on disease greater than if the malformation did not exist. Union of the different curtains of the tricuspid valves is found not infrequently in autopsies, and in a certain proportion of these cases, may be due to disease of intra-uterine life. The proportion of cases in which the lesion dates from a period anterior to birth, is undoubtedly greater in the instances of union of the curtains of the tricuspid than of the mitral valve. It appears that the difference in tendency to valvular disease between the two sides of the heart, which is so marked after birth, is reversed during fœtal life; in other words, the tricuspid valve is as much more likely to take on disease before, as the mitral valve after birth.

To determine in early life that malformation of some kind exists, is usually not difficult, provided the abnormity be of a nature, and sufficient in degree, to induce marked disorder. Palpitation, dyspnœa, or cyanosis, existing from birth, or developed shortly afterward, and either persisting or recurring more or less frequently, point to a congenital difficulty. To determine the particular kind of malformation, however, is a problem in diagnosis by no means always easy. With reference to the latter discrimination, it is important to bear in mind that of a given number of malformations, after the age of twelve years, in a very large proportion there exists contraction at the pulmonic orifice. Of thirty-nine cases analyzed by Dr. Peacock, obstruction to the passage of blood into the pulmonic artery existed in thirty-two. It is also to be kept in view that in a very large proportion of the instances in which obstruction at the pulmonic orifice exists, either there is patency of the foramen ovale, or deficiency of the auricular or ventricular septa, or both. If pulmonic obstruction be determined, the chances are that the last mentioned malformations coexist. Guided by the law of probabilities, if a person survive several years with manifestly some cardiac malformation, we shall seldom err in presuming that there exists pulmonic obstruction. But physical signs may convert this presumption into a conclusion quite positive. A bellows-murmur referable to the pulmonic orifice points to this as the seat of an abnormal condition. We have seen that a systolic murmur may be referred to the pulmonic orifice. The maximum of the intensity of the murmur will be at the base of the heart on the left side of the sternum, or the murmur may be limited to that situation. It is not propagated into the carotids. Attention to the pulmonic second sound of the heart may afford additional aid in the diagnosis, this sound being found to be abnormally weak or wanting. In connection with a murmur thus localized, in a large proportion of cases there will be present the physical evidence of enlargement of the right ventricle. A diastolic murmur referable to the pulmonic orifice, or a pulmonic regurgitant murmur, may be discovered, especially if the right ventricle be hypertrophied. I have lately met with such a murmur distinctly appreciable over the body of the heart, on the right side of the sternum, and at the xiphoid cartilage. Communication of the two ventricles through an aperture in the septum, gives rise to a systolic murmur. A murmur thus produced will not be propagated either along the course of the aorta or pulmonic artery, and will have its maximum at or near

the base of the heart. By these points its source may be determined with considerable confidence, but not with positiveness, for, exclusive of malformations, intra-ventricular murmurs are occasionally incident to disease which cannot be traced either to the arterial or auriculo-ventricular orifices by the ordinary rules of localization. The passage of blood through an open foramen ovale probably rarely, if ever, gives rise to a murmur.[1] The clinical study of cases of malformation, with respect to the physical signs, is highly interesting, and claims more attention than it has as yet received.

Of the causes of death in the various forms of malformation, the most frequent are, 1st. Cerebral disturbance resulting from the defective aeration of the blood and congestion of the brain; and, 2d. Imperfect expansion, collapse and engorgement of the lungs. It is worthy of note that dropsical effusions, so common in lesions of the heart originating after birth, occur less frequently than would be expected from the obstruction to the circulation incident to many of the malformations. Death occurs not very infrequently from tuberculosis in the cases in which life is prolonged for several years. Of 56 cases, analyzed by Dr. Peacock, in which patients affected with different forms of malformation survived the age of eight years, in 9 tuberculosis became developed, being a ratio of 16.07 per cent. In six of the nine tuberculosis cases cyanosis existed in a marked degree. This appears to militate against the incompatibility of tuberculous disease and venosity of the blood, as asserted by Rokitansky. It is, however, certain that diseases of the heart developed after birth, and phthisis are rarely associated; and the inquiry arises, whether there is a law here applicable to morbid conditions and not to malformations. This is a question to be settled by further statistical data.

The general principles of treatment in cases of malformation, may be embraced in a very few words. They relate to measures to protect against cold; avoidance of over-exertion and great mental excitement; together with such palliative measures as the particular circumstances in individual cases may indicate.[2]

[1] In six cases of open foramen ovale, reported by John W. Ogle, M. D., assistant physician at St. George's Hospital, London, no murmur was discovered during life.—*British Med. Journ.*, p. 500, 1857, from *Journal de la Physiologie*, etc., publié sous la direction du Docteur E. Brown-Séquard, Janvier, 1850.

[2] It is proper to state that the foregoing account of congenital affections has been mostly borrowed from an analytical review of Dr. Peacock's work, written by the author, and contained in the *American Journal of Medical Sciences*, No. for July, 1858.

CYANOSIS.

Blueness, or a purple color of the surface of the body and the mucous surfaces open to observation, occurring in connection with malformations of the heart, has been considered as constituting an affection called cyanosis,[1] morbus cæruleus, or blue disease. For the sake of precision, these names should be restricted, as they usually are, to the peculiar coloration due to abnormal conditions which are congenital, although this effect may not be manifested for some time after birth. But an analogous, if not identical appearance of the integument is observed in some cases of organic disease of the heart developed at different periods of life, and also independently of any cardiac lesions. It is well marked, for example, in the algid, or, as it is often termed, the cyanotic stage of epidemic cholera. This fact is to be borne in mind with reference to the rationale of the blueness which characterizes certain cases of cardiac malformation. The nature of the connection existing between cyanosis and malformations of the heart, has been much discussed, and is still open for discussion. To consider the subject at much length, would be inconsistent with the practical character of this work. I shall therefore present, very briefly, the views which seem to comport best with our present knowledge.

Cyanosis was attributed by Morgagni to congestion of the venous system caused by obstruction at the origin of the pulmonic artery. John Hunter attributed it to the admixture of venous and arterial blood in consequence of abnormal communication between the auricles or ventricles, or an abnormal arrangement of the primary vessels. The latter was the current doctrine until within the past few years the explanation of Morgagni has been revived and maintained by several distinguished pathologists—Louis and Valleix in France, Hasse and Rokitansky in Germany, Jay and Peacock in England, and the late Moreton Stillé, of this country.[2] Many distinguished pathologists, however, still adhere to the Hunterian theory, while some adopt both explanations, referring the affection in certain cases to venous congestion solely; in other cases, to the

[1] κύανος, blue, and νόσες, disease.

[2] On Cyanosis, or Morbus Cæruleus, by Moreton Stillé, M. D., *American Journal of Medical Sciences*, new series, vol. viii., 1844, p. 25.

admixture of the two kinds of blood, or to the combination of these two abnormal conditions.

In the endeavor to settle upon the true explanation of cyanosis, the first and most important point of inquiry is, whether it be uniformly associated with any particular class of malformations. This point is not readily ascertained, because, in the great majority of cases, malformation does not consist of a single abnormity, but several abnormal conditions are combined. Thus, obliteration or obstruction of the pulmonic orifice generally involves an open foramen ovale or deficiency of the ventricular septum. The former induces congestion of the venous system; the latter occasions admixture of the venous and arterial blood. Analyses of large collections of cases, in fact, show that, in by far the greater number, there exist pulmonic contraction, and, at the same time, communication between either the ventricles or auricles, or both. Of 62 of the cases collated by Stillé, in which the condition of the pulmonary artery was reported, in 53 it was obstructed or impervious. In the remaining 9 cases, the author concludes that the abnormal conditions present were of a nature to give rise to congestion of the venous system. On the other hand, in 5 only out of 71 cases collected by the same author was communication between the two sides of the heart wanting. Cyanosis has been observed when the foramen ovale was not open, and there was no deficiency of the ventricular septum, nor transposition of the primary vessels; and contraction of the pulmonic orifice is not always present. Again, cases have been reported in which the two kinds of blood must have been very freely mixed, as in some instances in which there existed a single ventricle, without cyanosis; and cases of great congenital pulmonic obstruction have been observed without cyanosis.

In short, constancy of connection with any particular class of malformations is not, as yet, established. Cyanosis cannot be considered as having any fixed special anatomical character. It may be associated with numerous and different abnormal conditions.

Continuing to regard the different forms of malformation as giving rise either to venous congestion or admixture of the two kinds of blood (although, as has been seen, both effects are usually combined), the facts adduced by Stillé, Peacock, and others, appear to show conclusively that the former effect is concerned in the production of cyanosis much oftener and to a much greater extent than the latter. The exceptions to the rule that obstruction either at the pulmonic orifice or elsewhere, inducing congestion of the

veins and venous radicles, exists in cases of cyanosis, must be exceedingly infrequent, if, indeed, there are any exceptions to the rule, and the instances in which great congenital obstruction at the pulmonic orifice are not attended by cyanosis, may, perhaps, be explained, as suggested by Dr. Peacock, by supposing that the right ventricle becomes, under these circumstances, sufficiently hypertrophied to compensate for the obstruction by the increased power of its contractions. The researches of Stillé have sufficiently established the fact, already stated, that the most complete commingling of arterial and venous blood, either by direct communication between the two sides of the heart or by mal-arrangement of the vessels, is not always adequate to give rise to cyanosis; and that, as regards intensity, cyanosis bears no constant relation to the freedom of communication between the two sides of the heart or the different systems of vessels. But the establishment of these facts does not prove that the commingling of the two kinds of blood is never involved in the production of cyanosis. That in certain cases this is an important element is probable. It is evident that the coexistence of pulmonic obstruction with either an open foramen ovale or deficiency in the ventricular septum must contribute in no small measure to the admixture of the blood through these communications; and hence it is intelligible that when these malformations are combined (as they usually are), cyanosis is much more likely to be the result than when either exists independently of the other.

The general conclusions, then, most consistent with our present knowledge of the subject are that cyanosis involves, in the vast majority of cases, if not invariably, venous congestion due to contraction or obliteration of the pulmonic artery, or to some other malformation which occasions obstruction to the flow of blood from the systemic veins; that it may be produced by obstruction alone without any admixture of the arterial and the venous blood, but that the latter may contribute, more or less, to its production. The presence of venous blood in the arterial system, it is to be remarked, contributes, not alone by the purple color which it acquires from the admixture to the cyanosis, but by increasing the venous congestion. The capillary circulation is impeded, and the flow of blood through the veins retarded in proportion to the venosity of the arterial blood.

The blueness of the skin in cyanosis is due, of course, to the blood contained in the minute or capillary vessels. Now, inasmuch

as obstruction of the venous system occurs, frequently in a great degree, in cases of organic lesions of the heart arising from disease developed after birth, the question arises, why is it that cyanosis is peculiar to, or at least occurs so much oftener and to a greater extent in connection with congenital affections? It is highly probable that the answer to this inquiry is contained in a suggestion by Dr. Chevers,[1] viz., that the capillary vessels become much more largely expanded when obstruction to the circulation exists before birth, or prior to the full development of the body, the vascular system being more readily dilatable, than in the adult. Cyanotic phenomena, however, are not exclusively observed in connection with malformations. They may be developed at any age as a result of obstruction at the right side of the heart in conjunction with deficient aeration of the blood. They are seen in cases of pulmonary obstruction due to atelectasis, collapse of lung, capillary bronchitis, etc. They are well marked, as already stated, in the blue stage of epidemic cholera, being dependent, in the latter affection, in a great measure, on capillary congestion proceeding from the abnormal condition of the blood itself. The appearance of the tegumentary surfaces in these various affections does not differ essentially from that in cyanosis, the main difference being that the blueness or lividity is very rarely, if ever, so extreme as in the cases in which it is dependent on congenital affections.

The discoloration in different cases of cyanosis differs greatly in degree. Between slight blueness and darkness approaching nearly to blackness, in a sufficient variety of cases, every degree of gradation will be manifested. All portions of the body are not alike affected. Certain parts, viz., the lips, around the eyes, the cheeks, the ears, the extremity of the nose, the roots of the finger nails, and the genital organs, present a change in color more marked than over the surface generally. The blueness may be limited to parts in which the skin is delicate and the capillary vessels abundant. The degree of discoloration varies also greatly at different periods in the same case. Its intensity is increased by fits of coughing, muscular exercise, mental emotions, and any cause which excites the action of the heart. The cyanosis may exist only under these circumstances, being absent when the heart is tranquil. It is always increased by any intercurrent pulmonary or cardiac disease.

[1] Dr. Peacock, op. cit., p. 128.

Although dependent on malformations, cyanosis is not always manifested at or immediately after birth. Of 71 cases analyzed by Stillé with respect to this point, it was congenital in 40, and occurred in the remaining 31 cases at various periods after birth. It may not occur until several years after birth. When this is the case, it is reasonable to presume that some disease of the heart or lungs has been added to the malformations, increasing the venous obstruction occasioned by the latter. It has been observed to follow a blow on the chest. The development of cyanosis after birth has been accounted for by supposing that in these cases a communication between the two sides of the heart either takes place or is enlarged at the time when the cyanosis occurs. Rupture or perforation of the foramen ovale may happen after birth, or the size of an existing aperture may be increased. The same may be said with regard to the interventricular septum at the undefended space. This explanation is based on the supposed importance of the admixture of the venous and arterial blood in the production of cyanosis. That it is applicable to certain cases is not improbable. On the other hand, cyanosis in some instances exists at birth and afterwards diminishes. It may even disappear; but such cases must be extremely rare.

Although cyanosis is regarded as a distinct affection, it is sufficiently evident that it is only a symptom of certain congenital affections of the heart. It has no claim to be considered as an individual disease. It is associated with other symptoms of malformation, viz., palpitation, dyspnœa, etc. When present, habitually, in a marked degree, the patient generally is remarkably susceptible to cold, and the temperature of the body is lowered. The muscular power is deficient. The muscles do not attain to a full development. The faculties of the mind are also often imperfectly developed and feeble. Enlargement of the pulpy extremities of the fingers, with incurvation of the nails, constituting what is called "clubbed fingers," is observed in some cases. I have met with this charge in a marked degree, in connection with organic lesions of the heart occurring after adult age, not associated with tuberculosis of the lungs.

The diagnosis rarely involves much difficulty. Discoloration of the surface, either general or partial, present habitually, or occurring whenever the action of the heart is excited; existing at, or developed shortly after birth in the great majority of instances; accompanied by palpitation, dyspnœa, tendency to syncope, etc., either constantly or in paroxysms; muscular weakness, abnormal

coolness of the surface and susceptibility to cold; these are diagnostic points pertaining to the symptoms. In addition to these points, physical signs denoting malformation of the heart are generally determinable, consisting of those which denote enlargement of the organ, together with organic murmurs, the latter being often referable to the pulmonic orifice. The lividity due to certain pulmonary affections in children, is to be discriminated by the previous history, taken in connection with the presence of symptoms and signs pointing to the lungs as the seat of disease, and the absence of the symptoms and signs of malformation of the heart.

The prognosis in cases of malformation of the heart accompanied by cyanosis, is unfavorable. If the discoloration be congenital, intense, and persisting, it denotes a condition of the heart which is generally incompatible with a duration of life beyond a few weeks or months. If moderate or slight, or occurring only in paroxysms, patients sometimes live for many years, and even long life is possible. The statistics collected by Stillé with regard to the duration of the disease, show, at a glance, the diversity of cases in this respect. Of 40 cases, in all of which the cyanosis was congenital, death occurred within 23 days after birth in *seven;* between 23 days and 10 weeks, in *three;* between 10 weeks and 1 year, in *seven;* between 1 year and 10 years, in *ten;* between 10 years and 20 years, in *ten.* Of these 40 cases life was prolonged to 29 years, to 35 years, and to 57 years, respectively, in a single instance.

The treatment of cyanosis resolves itself into that of malformations of the heart. The few remarks already made comprise all that it is necessary to say under this head.

CHAPTER VI.

CERTAIN AFFECTIONS INCIDENTAL TO ORGANIC DISEASES OF THE HEART.

Formation of Clots and Fibrinous Coagula in the Cavities of the Heart.—Clots formed after death and at the close of life—Fibrinous coagula formed during life—Their pathological connections—Their formation in organic affections of the heart—Symptoms denoting their formation—Physical signs and diagnosis—Prognosis—Treatment.

Polypi of the Heart.

Angina Pectoris.—Symptoms characteristic of—Description of paroxysms—Exciting causes—Pathological character and relations—Infrequency of the affection—Influence of age and sex—Gravity and prognosis—Diagnosis—Treatment.

Enlargement of the Thyroid Body and Prominence of the Eyes.—Phenomena descriptive of the enlargement of the thyroid body—Morbid appearances of the heart in fatal cases—Cases observed by the author—Supposed pathological connection with excessive action of the heart—Phenomena descriptive of the prominence of the eyes—Different explanations—Diagnosis—Prognosis in cases of enlargement of the thyroid body and prominence of the eyes—Indications for treatment.

Reduplication of the Heart-Sounds.—Different varieties of reduplication and their relative infrequency—Cases of reduplication of both sounds—Cardiac lesions found after death in cases of reduplication—Mechanism of reduplication—Bearing of the facts pertaining to reduplication on the mechanism of the normal heart-sounds—Mode of distinguishing the different varieties of reduplication—Pathological import and diagnostic significance of reduplications—Treatment.

The caption to this chapter includes several pathological events which are liable to occur in cases of organic disease of the heart, but which do not belong exclusively to the clinical history of any particular lesions. They occur in different forms of organic disease, and all of them do not involve, of necessity, the existence of an antecedent structural lesion. Hence, although these events are quite dissimilar in character, they may conveniently be grouped together. The first of these events which will be considered is the formation of clots and fibrinous coagula within the cavities of the heart; the second is the occurrence of pain and other symptoms in paroxysms, commonly known as angina pectoris; enlargement of the thyroid body and prominence of the eyeball will be next noticed, and, finally, reduplication of the heart-sounds. These subjects will be treated of only so far as, with our present knowledge, they are of interest and importance in a practical point of view.

FORMATION OF CLOTS AND FIBRINOUS COAGULA WITHIN THE CAVITIES OF THE HEART.

The cavities of the heart are usually found to contain, after death, coagulated blood, or clots, in more or less abundance. These are found oftener and in greater abundance in the right auricle and ventricle than in the cavities of the left side of the heart. This is owing to the fact that, at the time of death, the cavities of the right side of the heart contain, in general, a much larger quantity of blood than the left auricle and ventricle. Other things being equal, the size and number of clots will be proportionate to the amount of blood remaining in the heart-cavities after life has ceased. The clots to which reference is now made are formed *post-mortem.* The blood in the cavities coagulates after death, as it does when drawn from the vessels by venesection during life. These clots are variable as regards size, form, consistence, and color. They are sometimes uniformly dark and friable. In other instances they are more resisting, but never extremely dense, and present, on the surface extending more or less over the periphery, a layer of fibrin devoid of red globules, or hæmatin. The latter is identical with the buffy coating of blood coagulated, in certain cases, after venesection. It is sometimes tolerably firm, and, in some instances, probably from the imbibition of serum, it is of a soft, jelly-like consistence. A distinctive feature of the clots now referred to is, they are loose, *i. e.*, not attached to the endocardium, and not strongly intertwined with the tendinous cords or fleshy columns. They may extend from one cavity to another through the auriculo-ventricular orifices, and into the large vessels, the arteries and veins, connected with the heart. It is not uncommon to find prolongations of considerable length contained in the large arteries, especially the pulmonic artery, consisting of fibrin, more or less solid, and colored, to a greater or less extent, by the presence of red globules. The occurrence of *post-mortem* clots undoubtedly depends, in a great measure, on the condition of the blood. They are more likely to be formed in those diseases in which the fibrinous constituent of the blood is in excess (hyperinosis); and, under these circumstances, the proportion of colorless fibrin in the clots will be increased. On the other hand, after

certain fatal affections, as is well known, the blood coagulates imperfectly, and sometimes not at all, the cavities of the heart being filled with blood entirely liquid.

To the clots just described the older pathologists attached much importance. They were regarded as *ante-mortem* productions, and included in the class of the so-called *polypi* of the heart, being supposed to give rise to a multitude of symptoms during life, and to be frequently the cause of death. That they are formed after death is certain, but the question arises, whether they may not sometimes be formed during the last moments or hours of life, and, in fact, prove the immediate occasion of the cessation of the circulation. It is difficult, and indeed impossible, to settle this question demonstratively, but the affirmative is highly probable. That coagulation does take place before death in certain cases, and arrests the circulation, is not to be doubted. The coagula that are indubitably of *ante-mortem* formation will be presently considered. The question now relates to clots, loose or unattached, and not differing from those which are due to coagulation after death. It may be readily conceived that in certain diseases of the heart, and in various affections exclusive of these, at the close of life, when the circulation becomes so enfeebled that the blood accumulates and remains nearly stagnant in the cavities, coagulation may take place, and, to quote the language of Prof. Meigs, "the last fatal blow is struck by the formation of a heart-clot of greater or less size." The distinguished author just named accounts in this way for sudden death, in some puerperal cases, during syncope induced by assuming suddenly the erect posture, when recent delivery has been accompanied or followed by a large amount of hemorrhage.[1] The explanation is, to say the least, plausible; and its extension, by the same author, to account for the final cessation of the circulation in various chronic and acute diseases, is not irrational. This, however, can only be a matter for conjecture, since the clots found in the cavities of the heart do not differ from those which are formed by coagulation after death.

Masses of considerable size, consisting of coagulated fibrin, are often found in the cavities of the heart in post-mortem examinations which furnish intrinsic evidence of having been formed during life. This evidence consists in their density, the absence of

[1] *Vide* paper by Prof. C. D. Meigs, in the Philadelphia Medical Examiner, March, 1849. Also treatise on Obstetrics.

red globules, intertwining with the tendinous cords and fleshy columns, adhesion to the endocardium, grooving of their surfaces by the currents of blood, and certain changes due to molecular disintegration or decomposition. These characters denote that they are not produced by coagulation after death, nor as the final event in the act of dying, although they frequently prove the immediate cause of the arrest of the circulation. The date of their formation may be days and possibly weeks anterior to death. These coagula differ greatly in size and configuration. They may be formed in the cavities of the left, as well as in those of the right side of the heart, but more frequently in the latter, and oftener in the auricle than in the ventricle. They are frequently connected with the tendinous cords or fleshy columns, with which they are often very closely and strongly intertwined, the latter fact being alone sufficient to show their ante-mortem formation. The play of the valves, when coagulation is taking place, causes the fibrin to adhere to these parts, precisely as it is collected for experimental purposes, by whipping with a bundle of small sticks blood drawn from the body. By this whipping process the red corpuscles are expelled, and the coagula consist of pure fibrin. They are sometimes closely adherent to the endocardium, but it may be doubted if this ever occurs by means of an organized attachment. In the instances in which they seem to be grafted into the heart, the coagulated fibrin is probably deposited on an organized exudation or morbid growth. The opinion held by Hope, and even by some eminent pathologists of the present day, that masses of coagulated fibrin may become organized, increase by a process of growth, and undergo transformations of texture dependent on abnormal nutrition, must be considered untenable. In the heart, as elsewhere, the fibrinous element of the blood, whenever isolated and solidified, becomes, virtually, a foreign substance incapable of organization. The fibrinous masses are sometimes found to contain collections of liquid, varying in color and consistence, presenting an appearance of unilocular or multilocular cysts. They have been said to contain pus and softened tuberculous deposit. The latter statement has not been substantiated by adequate examinations. Either the liquefied portions have been imbibed from without or they are due to disintegration or decomposition commencing within the solid masses; and although these portions may present the gross appearances of purulent or tuberculous matter, the microscopical characters of the latter are wanting.

In this account of fibrinous coagula, reference is had to the formation of masses of considerable size, attributable to coagulation. They are to be distinguished from the deposits due to exudation, in other words, concretions of lymph, the size of which may be increased by layers of fibrin, constituting the vegetations or excrescences so often found attached to the valves and orifices of the heart. These have been already noticed in treating of valvular lesions, and they will be again considered in connection with endocarditis. They differ from the formations now under consideration in this respect, viz: they occur almost exclusively in the cavities of the left side of the heart, and more especially in the left ventricle.

Fibrinous coagula occur in various pathological connections, some of which are, as yet, imperfectly understood. They occur as a result of the accumulation and stagnation of blood in the cavities of the heart. So far, the conditions involved in their formation are mechanical. Conditions pertaining to the blood itself favor, and may be sufficient for their formation. One of these conditions is an inordinate proportion of fibrin, either from its being positively increased, or relatively, in consequence of the other constituents of the blood being diminished. Hence, fibrinous coagula are liable to occur in certain affections characterized by the excess of fibrin in the blood, especially when the mechanical conditions are combined. This combination exists especially in pneumonia, and in a large proportion of the fatal cases of this disease, coagula are found which must have been formed during life.[1] It also exists in cases of death after excessive loss of fluids, as in epidemic cholera. Inflammation of the endocardium disposes to coagulation, partly from the presence of deposits of exuded lymph, and perhaps, also, as stated by Rokitansky, from the contamination of the blood in consequence of the admixture of the inflammatory products carried into the circulation. If this latter statement be correct, it is intelligible that a similar contamination from the products of inflammation derived from other situations than the heart, may lead to the same result. The formation of fibrinous coagula is by no means limited to cases in which the heart is diseased. Some years since

[1] *Vide* Traité Pratique de la Pneumonie, par Grisolle. Grisolle establishes by comparative observations, that dense fibrinous coagula, adherent or closely intertwined with the tendinous cords or fleshy columns, are not only often found after death in cases of pneumonia, but rarely in fatal cases of typhoid fever, peritonitis, eruptive fevers, and cerebral maladies. Op. cit., 1841, p. 70 *et seq.* See, also, Richardson's prize essay On the Cause of the Coagulation of the Blood, London, 1858.

a heart was presented to me by my friend, Dr. C. H. Baker, in which the curtains of the tricuspid valve were literally tied firmly together by a mass of dense, colorless fibrin, portions of which were closely intertwined with the tendinous cords and papillary muscles, a prolongation of the fibrinous mass extending into the right auricle. The obstruction of the right auriculo-ventricular orifice was complete. The person had been in ill health, but complaining of no definite ailments, and not under medical treatment. He was found dead in bed, and an examination made under the direction of the coroner disclosed no other cause of death than that stated. The heart, exclusive of the fibrinous mass mentioned, was devoid of morbid appearances. It was not enlarged, and the valves were sound. The various pathological conditions, irrespective of those pertaining to the heart, under which fibrinous coagula are formed, constitute a field for clinical study, which claims more attention than it has as yet received. I shall content myself with this remark, and proceed to consider the formation of coagula as incidental to organic affections of the heart.[1]

The conditions derived from organic lesions of the heart, under which fibrinous coagula are liable to occur, are mainly mechanical, consisting of the accumulation and stagnation of blood within the cavities. The latter effects, as has been seen, follow obstruction of the orifices and dilatation with great weakness of the organ. In cases of mitral obstruction which has eventuated in dilatation of the right side of the heart, the time arrives, if life be sufficiently prolonged, when the right ventricle and auricle are constantly distended, the ventricular contractions being so feeble as to propel but a small quantity of blood into the engorged pulmonary vessels. Under these circumstances, the fibrin may coagulate, becoming adherent to the tendinous cords and fleshy columns, interfering with the action of the tricuspid valve, obstructing the auriculo-ventricular orifice, and thus prove the immediate cause of death. Or, if there exist aortic lesions involving obstruction or regurgitation, the left ventricle, after a time, reaches the limit of hypertrophic enlargement, and dilatation predominates, with consequent weakness, and inability to expel but a small part of the contents of this cavity. The same result may take place here, but it occurs more infrequently than in the cavities of the right side. Extrinsic

[1] The prize essay by Dr. Richardson may be consulted with advantage on this subject.

causes, which weaken the heart, and certain states of the blood favoring coagulation, will contribute to, and may suffice for the occurrence of this accident. The formation of coagula may lead rapidly to a fatal termination. In a certain proportion of the instances of sudden death, it is to be thus explained. In other cases, life continues for some time with an aggravation of all the symptoms referable to the heart, dating from the epoch when coagulation took place. The mechanism of the formation of coagula, under these circumstances, is analogous to that by which they are formed in sacculated aneurisms.

The symptoms of the formation of coagula in cases of organic disease of the heart are certainly not distinctive, but, taken in connection with all the circumstances in the case, they often point with much significance to this accident. The significance consists in the sudden and great increase in the intensity of all the symptoms referable to the heart, under circumstances which render this explanation reasonable, and when the striking change in the condition of the patient is not to be otherwise accounted for. The reasoning is by exclusion rather than by positive diagnostic evidence. The formation of coagula is to be strongly suspected if a person known to have an organic affection of the heart, which has eventuated in dilatation, be abruptly seized with notable increase of dyspnœa, amounting to orthopnœa, and persistent, with a distressing sense of oppression at the præcordia; the heart becoming irregular and tumultuous; the pulse correspondingly disordered, extremely frequent, and feeble; and the more remote symptomatic phenomena, such as lividity, dropsical effusion, coldness of the extremities, being aggravated in proportion. This suspicion is resolved into an opinion which may be entertained with great confidence, if a careful examination of the chest reveals no intercurrent pulmonary disease, nor a superadded cardiac affection adequate to account for the remarkable alteration which has ensued. An acquaintance with the symptoms and condition of the patient prior to the occurrence of the accident is important with reference to the diagnosis. If the case have not been previously under observation, the practitioner is not so well prepared to estimate properly the change as when it takes place under his own eyes. He can neither appreciate its extent nor its suddenness, and the latter is an essential point in the diagnosis. The practicability of the diagnosis presupposes that the coagula occasion obstruction to the circulation in consequence either of the space which they occupy, their situation at or near

the orifices, or their interference with the proper play of the valves. A mass of fibrin, of considerable size, situated in the auricular appendix, or attached at the apex of the ventricle, may not give rise to a degree of disturbance greater than seems fairly attributable to the organic lesions, the existence of which has been ascertained. So, if the coagula are slowly formed, the symptoms are gradually developed, and the diagnosis, under these circumstances, is impracticable.

Physical signs furnish but little aid in the diagnosis. The presence of coagula may occasion an endocardial murmur, but, as a rule, it is wanting, probably in consequence of the enfeebled action of the heart. Moreover, a newly-developed murmur, produced within a ventricular, or, possibly, an auricular, cavity, can hardly be discriminated from pre-existing murmurs referable to the valvular lesions which are present in the great majority of cases. Theoretically, either the tricuspid or the mitral valvular element of the first sound would be expected to be impaired or lost, according as the coagulated fibrin may impede the play of the one or the other of the auricular ventricular valves. But weakness of the heart suffices to diminish or annul the first sound. In short, the diagnostic points in cases of fibrinous coagula incidental to organic disease of the heart must, in general, be derived mainly from the symptomatic phenomena, not from physical signs.

The diagnosis of the formation of coagula in other pathological associations, is free from some of the difficulties incident to their occurrence in connection with disease of the heart. For example, with the knowledge of the fact that in cases of pneumonia terminating fatally this accident is apt to occur, if, in the progress of that disease, the impulse of the heart and the pulse suddenly become extremely tumultuous, frequent, irregular, and accompanied by a degree of dyspnœa not explicable by the development of inflammation in another lobe, or any new condition referable to the lungs, the grounds for suspecting coagulation within the heart may be stronger than in some of the instances in which it occurs in cardiac affections, for the latter may have already induced great weakness and disordered action of the heart, with corresponding disturbance of respiration, etc. The change, as regards the symptoms referable to the heart, is not so striking in the latter case as in the former, and other explanations than the formation of coagula are less available. Moreover, assuming that, in the case of pneumonia, the absence of previous cardiac disease has been ascertained,

if the presence of a coagulum give rise to an endocardial murmur, at the same time that the striking symptoms referable to the heart occur, this constitutes strong evidence of the nature of the accident.

The prognosis in cases in which the symptoms denote the formation of fibrinous coagula is in the highest degree unfavorable. If death do not speedily follow, the utmost to be hoped for is that life may be prolonged for a few days or possibly weeks. There are scarcely any chances for improvement, and none for recovery. Bouillaud entertains the belief that the coagula are sometimes dissolved and disappear. The ground for such a belief is so small as to render it excusable to conclude that, in the instances in which this favorable termination has been supposed to take place, an error of diagnosis was committed. Correctness of diagnosis, in fact, is of advantage only in enabling the practitioner to decide that a fatal result is inevitable. As incidental to organic affections of the heart, however, it is to be borne in mind that in most instances the condition of the patient prior to this accident was hopeless. The effect of the latter, in general, is only to hasten the period of relief from the sufferings incident to incurable disease.

As regards treatment, it follows from the remarks just made that, after coagula have formed, palliative measures are alone indicated. These consist of remedies to relieve dyspnœa and præcordial distress, stimulants to maintain the action of the heart, and revulsive applications, such as fomentations, sinapisms, and stimulating pediluvia. The idea of giving remèdies with a view to dissolve the solidified fibrin is absurd. To prevent the coagulation of fibrin, when circumstances are present under which it may be expected to occur, is legitimate, and may be an important object of treatment. This object involves, in the first place, obviating as far as possible the accumulation of blood in the cavities of the heart by measures which have been already considered in connection with the treatment of valvular lesions. Sedative remedies, pushed to the extent of retarding and weakening unduly the muscular contractions of the heart, are objectionable, among other reasons, on the ground that they may favor coagulation. Digitalis is by some writers regarded as a dangerous remedy on this ground in cases of advanced organic or other disease in which the organ is already enfeebled. This remedy, however, is unattended by danger with proper care, if it be true that while it retards the movements of the heart, it does not diminish the muscular power of the organ. In the second place, it is not improbable that certain

remedies may favor the solubility of fibrin, and in this way prevent coagulation. Various alkaline remedies have been supposed to have this effect. If it be true that the fibrin is held in solution in the blood by the presence of ammonia, according to the late researches by Dr. Richardson, it would seem to be a rational inference that ammoniacal remedies must be the most efficient in fulfilling this second object in the prophylactic treatment.

POLYPI OF THE HEART.

The clots and fibrinous coagula which have been considered, were regarded, as already stated, by the older pathologists as morbid growths resembling the polypi met with in the uterus, nasal passages, and other situations. They were called *polypi* of the heart, and the term *polypoid* formations is still very generally applied to them. It is needless to say that, pathologically, they bear no resemblance to polypi, since they are not morbid growths, and never become organized. They cannot, therefore, with propriety be said to be *polypoid*, and the use of this term has been designedly avoided in the foregoing remarks. Abnormal productions, however, may occur within the cavities of the heart, which are analogous to polypi or polypoid growths. Grisolle[1] gives the results of the analyses of seven cases, which he states to be all the authentic cases on record.[2] In nearly all these cases there existed a pedunculated tumor varying in size from a pigeon's egg to a hen's egg; in six of the cases contained in the right, and in one instance in the left auricle. In all of the cases in which the point of attachment was indicated, it was at or near the foramen ovale. In four cases the tumor extended through the auriculo-ventricular orifice into the ventricle. The peduncle was formed apparently by the endocardial membrane which generally extended over the tumor. The form of the polypi was pyramidal, and they presented in some instances a smooth, and in other instances a lobulated

[1] Traité de Pathologie Interne, 1852, tome second, p. 389.

[2] The museum of the Boston Society for Medical Improvement contains a specimen presenting a tumor hanging loose in the cavity of the left auricle, supposed to be malignant from the coexistence of malignant disease in the lung and about the elbow. *Vide* printed Catalogue, 1847, p. 88.

surface. The substance of the tumors varied in appearance. In one case it had a fleshy aspect, in one case it resembled a fungous growth, and in two cases the texture was fibrous. In every case there was hypertrophy of the auricle and the corresponding ventricle.

The formation of true polypi in the heart differs from that of clots and fibrinous coagula in this, viz., it goes on slowly, and considerable time must be required for the growth of a tumor of sufficient size to occasion serious inconvenience. Hence, the symptoms are developed gradually and imperceptibly, not abruptly as in cases of coagula. When developed, the symptoms denote an organic affection of the heart, without pointing to the existence of a tumor. The physical signs, as well as symptoms, are not distinctive of the nature of the affection. They may indicate obstruction, or regurgitation, or both, associated with more or less cardiac enlargement. Taking into view the excessive infrequency of these growths, their existence can hardly be suspected from the phenomena during life.

It is needless to consider the treatment. The fact of some obscure cardiac affection being determined, the indications will be derived from the condition of the heart as respects enlargement, and the symptoms.

ANGINA PECTORIS.

An extremely distressing and grave affection, occurring, happily, in a very small proportion of cases of organic disease of heart, is commonly known by the name of angina pectoris.[1] This name was applied to the affection by Dr. Heberden, who was the first to give a full and clear description of it in 1768. The affection is characterized by paroxysms of intense pain emanating from the neighborhood of the præcordia, extending thence in various directions, often into the left shoulder and down the arm, accompanied by indescribable anguish, a sense of suffocation, and a feeling of impending death. These are symptoms characteristic of the affection in a severe form. The præcordial pain is variously described by patients, being lancinating, contusive, lacerating, burning, or con-

[1] ἄγχω, to strangle.

strictive. Its centre, or focus, generally appears to be over the heart, to the left of the sternum. It is sometimes most intense beneath the sternum; and Valleix cites two instances in which the greatest intensity was referred to the right of the sternum.[1] The pain radiates, as it were, into both sides of the chest, into the back, extending, as already stated, often into the left upper extremity, but sometimes, as in one instance under my observation, into both upper extremities, and occasionally into one or both lower extremities. Dr. Hope met with several instances in which it pervaded all the extremities. Not uncommonly it ascends to the neck in front or behind, and I have met with an instance in which it extended to the jaws and temples. The pain in the upper extremity sometimes appears to end abruptly at the shoulder, and in other cases at the elbow. I have known it to be felt acutely in the forearm, and not in the arm or shoulder. Not infrequently it seems to follow the course of the nerves, and is felt over the whole affected extremity, even to the fingers. The pain is attended by a feeling of numbness, or as if the limb were paralyzed. Numbness referred to the testes is mentioned by Dr. Walshe as a rare concomitant. Tenderness to the touch, or hyperæsthesia of the integument over the situations in which the pain is felt, has been observed in some cases, especially in females.

The affection is essentially paroxysmal. The patient is seized suddenly, often when in motion, and the paroxysm has been repeatedly noticed to occur in walking up an acclivity, after a meal, and especially against a strong current of air. Instant and complete rest is imperative. He seizes hold of some firm support, if any be at hand, or he finds it necessary to take a sitting or recumbent posture, which he does with great caution, and remains as immovable as possible until the paroxysm passes off. The pain is by no means the sole element of the distress. The sense of suffocation and of impending dissolution occasions hardly less suffering. There is, in addition, often a feeling of anguish which patients find it impossible to describe. Dyspnœa is not a constant element of the paroxysms. It may be present, but is frequently wanting. The respiratory movements are often momentarily suspended or restrained by an act of the will, from fear of increasing the pain and distress, but the ability to expand the chest and breathe regularly is not necessarily impaired. An intelligent patient recently

[1] Guide de Médecine pratique.

under my observation, who was subject both to angina and paroxysms of dyspnœa, described them as clearly distinct from each other, nor were the two liable to occur at the same time. Not infrequently the respirations continue unaffected during the paroxysms of angina, and they are rarely more than moderately accelerated. Palpitation is often present. It is rare for the action of the heart to be undisturbed, certainly if the paroxysms are severe. Often the heart acts with great violence, seeming, to quote the language of a patient, "as if it would leap out of the mouth." It is frequently irregular and intermitting, the pulse sometimes indicating vigor, and in other instances feebleness, of the ventricular contractions. The pulse, however, has been observed to be unnaturally slow during the paroxysms. The countenance is pallid, and expresses great anxiety and distress. The change in this respect may be very striking, a deathlike complexion, with great haggardness of the features, suddenly taking the place of an appearance of health. Lividity is occasionally observed. The surface is cold, and frequently bathed in perspiration. The faculties of the mind remain unaffected. A free secretion of limpid urine takes place in some cases. The reader who has not witnessed a severe paroxysm, may form from the foregoing account some idea of the distressing nature of this affection. There are few, if any, diseases which give rise to greater suffering. A patient who experienced the excruciating torture of daily attacks for several months before he found relief in death, made a dying request that I should examine his body post-mortem, in the hope that something might be thereby ascertained which would lead to the means of relieving others in like manner afflicted; a request with which I did not fail to comply.

The paroxysms of angina, in different cases, differ much, not only in severity, but in their frequency of recurrence, their duration, etc. They do not always have the severe character which has been portrayed in the foregoing sketch. They are sometimes comparatively mild. The affection in some cases commences with mild paroxysms, which may progressively become more severe; but in other cases the first attack is intensely distressing. Their duration is extremely variable. Often they last only for a few moments, but in some of the instances which have fallen under my observation, the suffering has continued for several hours. The cessation of the paroxysm is frequently as abrupt as the commencement. I have known a laborer to be attacked repeatedly while at work, and, resting for a few moments till the paroxysm ceased, at once resume his labor.

In some instances, however, relief is gradual. There exists an equal diversity, in different cases, as respects recurrence of the paroxysms. They may recur after intervals of a few hours, or even moments; or days, weeks, months, and years may elapse between successive attacks. The paroxysms are apt progressively to become more and more frequent, as well as more and more severe. Patients sometimes have repetitions, more or less frequent, during several days or weeks, and a respite for several months follows. The affection pursued this course in one of the cases which I have observed. In the vast majority of cases in which a paroxysm has once occurred, other attacks follow after longer or shorter intervals. Clinical experience furnishes very little ground for encouraging patients to hope for future exemption; on the contrary, it is almost certain that the affection will continue, and become more serious. I have met, however, with one instance in which two extremely severe paroxysms occurred, the interval being about forty-eight hours, and each paroxysm lasting from three to four hours, in a patient aged sixty-seven, who subsequently never had a recurrence, dying, after ten or twelve years, with symptoms of cardiac disease.

The paroxysms of angina frequently appear to be induced by some exciting cause, such as ascending an elevation, muscular exertion of any kind, mental excitement, etc. But in many instances they occur without any such cause, taking place in the night, or when the person affected is perfectly at rest in body and mind. When they recur with great frequency, slight causes seem adequate to determine an attack. I have known the act of swallowing solid food sufficient, so that the patient resisted as long as possible the desire to take nourishment. In the same case the paroxysms often occurred during sleep, appearing to the patient to be produced in consequence of dreams. On account of this liability, he hardly dared to sleep. The suffering in this case was beyond description. In some of the cases which I have observed, the paroxysms occurred only when it seemed fair to refer them to some obvious exciting cause. In another case, to which I have before alluded, the paroxysms always occurred, as it were, spontaneously, and causes which provoked severe attacks of dyspnœa, or cardiac asthma, never occasioned angina. Cardiac asthma, according to Dr. Stokes, is frequently confounded by practitioners with angina, but, certainly, these two incidental affections are sufficiently distinct, although they may both occur in the same case, and, perhaps, simultaneously in some instances.

We come now to inquire what is the pathological character of this affection, and what are its pathological relations. The points involved in these inquiries have been much discussed, and pathologists are by no means agreed respecting them. Without canvassing different hypotheses, it seems to me sufficiently clear that the affection must be considered as a form of neuralgia, or, at all events, that the painful element in the paroxysms is essentially neuralgic. The various kinds of pain are those which belong to neuralgic affections. The radiation of the pain in different directions is characteristic of the latter. Moreover, in some instances the pain distinctly follows the course of the nerves of the extremities. The identity is further shown by the abruptness of the attack, the suddenness with which the paroxysm frequently ends, and the completeness with which it disappears, leaving no trace of the affection except a certain amount of soreness and prostration. Dr. Heberden and others have attributed the pain to spasm; but a spasmodic contraction of the heart sufficient to occasion such prolonged as well as intense suffering would be incompatible with life. Disturbance of the muscular action of the heart is not, as a rule, proportionate to the amount of pain. The pain in some instances is extreme, while the movements of the heart are but little disturbed. The violent and disordered action which undoubtedly constitutes an element of the paroxysms of angina, may be said to be spasmodic, but in the same sense that functional disturbance, or palpitation, occurring under other circumstances, involves spasm. That this element is distinct from the painful element, is shown by the fact that functional disturbance of the heart, be it ever so great, is rarely accompanied by much pain. Neuralgic pain, and disturbed muscular action of the heart, are thus two elements of angina, which although, associated, are pathologically distinct. The term *cardialgia*, were it not appropriated to denote pain supposed to be referable to the cardiac extremity of the stomach, would be a more appropriate name than angina, for the affection under consideration. This term is applied by Forget to pain seated in the heart.[1]

As regards the pathological relations of angina, it involves, in general terms, the existence of some organic affection of the heart. If this rule be not invariable, the exceptions are so few that they may practically be disregarded. Of 45 cases in which the post-mortem appearances were recorded, collected from various sources,

[1] Op. cit.

and analyzed by Sir John Forbes in an essay published fifteen years ago,[1] the heart was reported to be unaffected in two instances only; and in view of the fact that certain morbid conditions of the heart have not been fully recognized until within late years, it must be considered as highly probable that in the two excepted instances, structural lesions may have been overlooked. Assuming the constancy of organic disease, does angina involve the existence of any particular kind of lesion? In answer to this question, dissections show that the lesions found in different cases are not uniform, and that they do not agree, invariably, in any one or more appreciable morbid alterations. The heart may or may not be enlarged. Valvular lesions are either present or wanting. Calcification of the coronary arteries is the only appreciable lesion in some cases, but in other cases these arteries have been found to be entirely healthy and free from obstruction. Fatty degeneration and softening are sometimes observed, but by no means as a rule. In the two most severe cases that have fallen under my observation, death being in both due to the angina, the substance of the heart was firm, presenting no appearance of fatty change. In short, angina has no settled anatomical character beyond the fact that it is incidental to organic disease of some kind. The affection occurs oftener in connection with lesions seated in the aorta than elsewhere. Aortic lesions were present in 24 of 39 cases analyzed by Dr. Forbes. Judging from physical signs, as well as the results of dissection, in the cases that have fallen under my observation, I should say that lesions of the aorta or the aortic orifice are present in much the larger proportion of instances. But that these lesions are not essential is sufficiently established. Doubtless some one or more particular abnormal conditions are common to all the cases of organic disease of the heart in which angina occurs. Logically considered, this is certainly more than probable. But the nature and seat of these conditions are yet to be determined. With our present knowledge we have nothing but conjectures on this point. There will be no advantage, in a practical point of view, in considering these. I will simply remark that the hypothesis which attributes the occurrence of angina to simple weakness of the heart, seems to me to be without any foundation. Not only is the heart weakened in a host of cases of dilatation and fatty degeneration,

[1] Cyclopædia of practical medicine, art. Angina Pectoris. I include in the number of cases of cardiac disease, those (four in number) in which obesity of the heart only existed.

without the occurrence of angina, but weakness by no means exists uniformly in the cases in which angina occurs. In several of the instances which have come under my observation, a paroxysm of angina was the first event to denote the existence of cardiac disease, and patients continue for a long time to take active exercise, performing, for example, severe manual labor, without any inconvenience save from the recurrence of angina. In the two severest cases that I have observed, the patients, even a few weeks before death, were confident of their ability still to work, if the frequent attacks could be prevented. These facts are inconsistent with much weakness of the heart. Moreover, the physical signs in certain cases, for a longer or shorter period, show that the heart acts with vigor, and, in some instances, with the abnormal power incident to hypertrophy.

According due weight to the facts just presented, angina pectoris is to be considered as a neuropathic affection, incidental exclusively to organic affections of the heart, and dependent on conditions pertaining to the different forms of cardiac lesions, which, it must be confessed, have not, as yet, been ascertained. With this view of its pathology, is it properly a distinct affection, or, in other words, an individual disease? Some writers are of opinion that it should be regarded merely in the light of a symptom.[1] It is symptomatic of cardiac disease, but not of any one of the varied lesions to which the heart is subject. It embraces a group of phenomena which are striking and distinctive. It derives from its peculiar features a strongly-marked individuality. It places the patient in great danger, irrespective of the lesions with which it is connected, so far as the latter are appreciable. It has, thus, all the attributes of a distinct affection, exclusive of the fact that it is secondary, *i. e.*, dependent on pre-existing disease, and this is true of not a few individual diseases.

Angina pectoris is a rare affection. Of over one hundred and fifty cases of organic disease of the heart, as evidenced either by the results of examination after death or well-marked physical signs, the histories of which are before me, this complication existed in seven only. This infrequency shows that it depends on conditions which, so far from being associated with structural lesions as a rule, are quite exceptional. Its connection with organic disease of the heart might be considered as merely a

[1] *e. g.* Bellingham, op. cit.

coincidence, were it not that it occurs only in this connection. With reference to the constant coexistence of organic disease, some writers, it should be remarked, hold to the opinion that the affection may occur independently of any cardiac lesions. The rare instances, reported many years ago, in which the heart was declared to be free from disease, have already been explained by supposing that abnormal appearances which had not then been studied were overlooked. Another source of error is in confounding with true angina certain neuralgic affections which simulate some of its phenomena. In certain cases of hysteria, for example, pain is referred to the præcordia, radiating, perhaps, in various directions, possibly extending to the left shoulder and arm, and accompanied by palpitation, but without the signs of organic disease. These are aptly styled by Dr. Walshe cases of pseudo-angina. They bear but a remote resemblance to well-marked cases of angina associated with cardiac lesions, and it seems warrantable, with our present knowledge, to limit the application of the term angina to the latter.

The affection is observed much oftener in males than in females. Of 88 cases analyzed with reference to this point by Dr. Forbes, 80 were males and 8 females. This immense disproportion shows that a very decided causative influence pertains to sex. A similar influence belongs to age. Of these 88 cases, in 72 the age exceeded fifty years. The rule with respect to sex, as well as age, however, it is to be borne in mind, is not without exceptions. I have met with the affection well marked in a female of twenty years of age, who subsequently died suddenly during an attack. It has been supposed to occur oftener in the higher than in the lower walks of life, but statistical data for this opinion are wanting.

It is a very serious affection, not only in consequence of the suffering which it occasions, and as denoting an organic affection of the heart, but, intrinsically, it involves great danger. In a large proportion of cases, death occurs suddenly, probably from arrest of the heart's action either by spasm or paralysis. This may take place in one of the early paroxysms. Instances of this kind are given by Dr. Latham.[1] But generally the paroxysms, becoming progressively more severe, recur with more and more frequency, and the fatal result is preceded by a period of intense suffering, varying greatly in duration in different cases. In many of these cases, the organic lesions, irrespective of the angina, are not such

[1] Lectures on Diseases of the Heart, Am. ed., 1847, p. 339.

as to be incompatible with a much longer duration of life. The complication of angina, therefore, in cases of cardiac disease, adds greatly to the gravity of the prognosis. It is, however, to be borne in mind that a patient may experience one or more severe attacks of this affection without further recurrence of the paroxysms. I have already referred to an instance of this kind which has occurred under my observation. There is some ground for hope, therefore, that the affection will not continue; but, it must be confessed, the chances against a favorable termination preponderate so vastly that such a hope is truly a forlorn one. Few affections are more indeterminate as regards duration than this. It may destroy life quickly, or after a lapse of time varying from a few weeks to many years.

The diagnosis of angina pectoris is, in general, easily made, provided the practitioner have a clear idea of its distinctive features. Its paroxysmal character, the attack generally being remarkably abrupt, and often ending as abruptly; the brief duration, in most instances, of the paroxysms; the intensity of the neuralgic pain, and its radiation in different directions, in most instances extending to the left shoulder and arm; the sense of suffocation; the feeling of approaching death; the indescribable anguish; the pallor, anxiety, and apprehension depicted in the countenance—these are diagnostic characters which, in well-marked examples, leave no room for doubt as to the nature of the affection. Paroxysms of dyspnœa or cardiac asthma, which are apt to occur in the progress of diseases of the heart, present points of difference so obvious that they need never be mistaken for paroxysms of angina. Their most prominent and distinctive feature, as the name dyspnœa implies, is difficulty of breathing. The sense of the want of breath is the chief source of suffering. The efforts of breathing are labored. The patient, instead of remaining perfectly quiet, is generally restless, frequently changing his position with the hope of finding relief. This difficulty of respiration does not belong to the history of angina. In the paroxysms of the latter the breathing is never extremely labored, and the patient often restrains voluntarily the respiratory movements, for fear of increasing his distress and sense of danger. Paroxysms of cardiac asthma rarely occur with the same abruptness as those of angina; nor do they end abruptly; and they are, as a rule, of much longer duration. They are rarely attended by acute pain. They are not characterized by that intense, undefinable anguish and feeling of approaching death which distinguish an attack

of angina. A patient suffering from dyspnœa, however intense, looks forward to relief and comparative comfort. A patient attacked severely with angina feels that he is momentarily in danger of death, and that, were the paroxysm to continue, he must inevitably die. In short, if these two affections are sometimes confounded, it is from inattention to the points involved in the differential diagnosis. It is not impossible, however, as already stated, for paroxysms of angina and of dyspnœa to occur in combination.

It is somewhat less easy to discriminate between true angina and certain neuralgic pains occurring under circumstances which cause them to simulate, to some extent, paroxysms of the former. Instances, however, in which there is much real difficulty must be very rare. The cases of pseudo-angina, to which reference has been already made, are observed in connection with hysteria, intercostal neuralgia, dyspepsia, anæmia, occasionally gout, etc. They are characterized by pain, more or less severe, in the neighborhood of the præcordia, extending thence in various directions, associated with functional disturbance of the heart's action, and the mental uneasiness and apprehension which the latter almost invariably occasions. These cases, exclusive of gouty subjects, occur, as a rule, prior to the age when persons are subject to angina. They are most apt to be met with among the young, and perhaps quite as often among females as among males. The acuteness of pain which characterizes attacks of angina, the anguish, and the sense of dying, are wanting, or, if the latter be present, it proceeds from the vague fear excited by unusual sensations, and does not consist in a feeling so intense and defined that it cannot be resisted. Pseudo-angina, moreover, lacks the abruptness of the beginning and ending of the paroxysms, as well as the brief duration, which characterize true angina in the great majority of cases. The associated disorders are different. Persons affected with pseudo-angina are hysterical, anæmic, dyspeptic, etc., and the nervous system is manifestly disordered. On the other hand, persons are often attacked with true angina when apparently in good health, and, at all events, the disorders just referred to are rarely found associated with it. The coexistence of organic disease of the heart is to be considered. In most instances of angina pectoris, cardiac lesions are determinable by physical signs; and, assuming, as has been done, that the affection always involves structural lesions of some kind, this is a point entitled to weight in the diagnosis. In cases of true angina we may expect to find, on exploring the chest, evidence of organic dis-

ease of the heart, and, in the majority of cases, signs of aortic lesions; in cases of simulated angina we may expect to find only functional disturbance of the organ. If the pain which bears some resemblance to that of angina be due to intercostal neuralgia, the latter affection is determined by finding tenderness on pressure limited to circumscribed spaces, by the side of the spinous processes of the vertebræ behind, in the intercostal spaces on the lateral surface of the chest, and near the median line in front, tenderness at these points being diagnostic of that affection. Finally, the question as regards the differential diagnosis between true and false angina can only arise when, if true angina be present, it is remarkably mild, and the instances in which this affection is not severe are very rare exceptions to a general rule.

Neuralgic pain is sometimes incidental to the various forms of organic lesion of the heart, without constituting angina pectoris. As a symptom of cardiac lesions, exclusive of angina, it is rarely present in a marked degree, and often wanting. Pain may be due to the coincidence of intercostal neuralgia, but in some instances it is evidently seated within the chest. Aside from other points, the absence of the strongly-marked paroxysmal character of angina suffices for the discrimination. There is no evidence that the existence of pain, or an occasional symptom of organic disease of the heart, denotes greater liability to paroxysms of angina than if this symptom were not present.

The treatment of angina pectoris embraces, 1st. The means to be employed to diminish the severity and shorten the duration of the paroxysms; and, 2d. Measures in the intervals to postpone or prevent the recurrence of the paroxysms.

The severity of the pain during the attack, and its neuralgic character, point to the propriety of opium; and clinical experience shows that this remedy is more efficient than any other, in affording relief, and bringing the paroxysm to a close. It is to be given in doses proportionate to the amount of suffering, and repeated after short intervals if the objects be not attained. The form of opiate selected should have reference to a prompt effect. Laudanum, the black drop, or an aqueous solution are preferable to the powder or solid gum, on account of the more speedy action of the former; but the salts of morphia are still more eligible in consequence of the ease with which they are given, and the greater certainty of their being retained. A convenient mode of administration is to place a grain upon the tongue. The succedanea of

opium, such as belladonna, hyoscyamus, etc., are not sufficiently effective. They may be employed, however, when the paroxysms recur repeatedly during the day, and it is not deemed judicious to continue to prescribe opium freely. Diffusible stimulants are to be given at short intervals. Brandy, or other kinds of spirit, may be employed; also the ethereal preparations and the carbonate of ammonia. Revulsive applications which act quickly, are indicated, viz., sinapisms, dry cupping, vesication with strong aqua ammonia, hot fomentations, and stimulating pediluvia. These several means are to be combined as convenience and the judgment of the practitioner may dictate in individual cases. The unexpected occurrence of the paroxysms and their brief duration, frequently render it impracticable to obtain medical aid before the attack has passed off. It is therefore important for the physician to give directions concerning the course to be pursued, in his absence, in the event of a recurrence of the paroxysms—an event to be expected, sooner or later, after an attack has been once experienced. Efficient treatment may often accomplish much toward lessening the intensity, and perhaps the continuance of paroxysms in certain cases. But when the liability to their recurrence is such that they succeed each other after brief intervals, and are produced by slight causes, palliative measures, which unhappily are all that can be resorted to, succeed but imperfectly. I have known the inhalation of chloroform to be employed in one case of this description, the severest case that has come under my observation, with marked relief; and, indeed, in that case, this after a time was the only palliative that could be relied upon.

Under the head of 'measures to postpone or prevent the recurrence of the paroxysms,' little is to be said. Rational treatment based on knowledge of the particular morbid condition or conditions involved in cardiac lesions, on which the affection is immediately dependent, cannot be laid down; for this knowledge is not yet acquired. Nor has clinical experience led to the discovery of the means of striking at the pathological root of the affection. There is no special medication to be pursued in cases of angina, with the hope of effecting a cure; and it is doubtful if any remedies exert a positive influence in lengthening the period of exemption from recurring attacks. Owing to the variableness of the intervals between the paroxysms, the latter effect may be imputed to remedies, when a protracted respite occurs merely as a coincidence. In a case in which the paroxysms had for some time recurred almost

daily, I was led to prescribe digitalis and hyoscyamus, which were taken in doses sufficient to affect the pulse distinctly and obscure the vision. The patient was free from the affection for three weeks. He imagined that he was cured, and was greatly elated. The paroxysms, however, returned, becoming more frequent and severe, and the case ended fatally in a few months. Here was apparently a temporary suspension of the malady as a result of certain remedies; but it is perhaps more reasonable to infer from the inefficacy of these remedies in other cases, that the prolonged interval after their use in this instance, was merely a sequence, not a consequence.

The treatment, after an attack of angina has occurred, with a view to postpone or prevent the recurrence of the paroxysms, resolves itself into, 1st. That indicated by the existing cardiac disease; and, 2d. Avoiding, as far as practicable, all exciting causes. Certain affections of the heart are always present, the existence of which was, perhaps, not known prior to the attack of angina. The nature and extent of these affections in different cases are to be determined as fully as possible with our present means of investigation, and that treatment pursued which would be indicated had the angina not occurred. It may reasonably be presumed that the treatment which is most judicious, in view of the lesions with which the heart is affected, will be likely to prove, in a greater or less degree, useful with reference to the recurrence of the paroxysms of angina. The indications falling under this head have already been considered in treating of the different forms of organic disease of the heart in previous chapters. The exciting causes to be avoided are those which experience shows are likely to occasion recurrence of the paroxysms. Strong mental excitement, violent muscular exercise of any kind, and especially walking rapidly, or ascending an acclivity against the wind, excesses in eating or drinking, etc., have been observed to bring on an attack. By scrupulously avoiding these and other exciting causes, which are peculiar to the individual experience of persons affected, it is highly probable that paroxysms may be warded off which would otherwise have occurred. Important, however, as is this part of the treatment, its effect is limited. Paroxysms often occur when they are not referable to any exciting cause. And when thus referable, it is probable that often the exciting cause (as the name implies) only determines the particular moment when the paroxysm takes place, anticipating somewhat the time when it would have occurred spontaneously.

ENLARGEMENT OF THE THYROID BODY AND PROMINENCE OF THE EYES.

The occurrence of enlargement of the thyroid body and prominence of the eyes, in certain cases of cardiac disease, may be conveniently noticed in the present connection, although, as will presently appear, it is by no means certain that these events necessarily involve the existence of any organic affection, and, indeed, that they depend on an abnormal state of the heart, cannot perhaps be considered as conclusively established. It is only within late years that the attention of clinical observers has been directed to these events, as connected with cardiac disease. The coincidence of enlargement of the thyroid body with affections of the heart was observed by Dr. Parry in seven cases.[1] A few instances had previously been reported. Subsequently, Dr. Graves dwelt upon this coincidence in his *Lectures on Clinical Medicine*, published in 1835, giving some cases that had fallen under his observation. Still more recently, the coexistence of prominence of the eyes, together with enlargement of the thyroid body, has attracted attention. Cases have been reported by Dr. McDonnell and Sir Henry Marsh, of Dublin, and by Dr. Stokes. The latter, in his late work on the *Diseases of the Heart and the Aorta*, devotes considerable space to the consideration of the subject. In our own country, Dr. Isaac E. Taylor has contributed an elaborate paper, containing an account of two cases which he has himself observed.[2] Dr. Begbie, of Edinburgh, and Robert Taylor, Esq., of London, have also reported cases. The latter gives the details of four cases which have fallen under his own observation, and an analysis, with regard to certain points, of twenty cases collected from various sources.[3]

The enlargement of the thyroid body is variable in degree, being considerable in some instances, but never so great as is often seen

[1] "Collections from the unpublished Medical Writings of the late Caleb Hilliard Parry, M. D.," London, 1825.

[2] New York Medical Times, vol. ii., No. 3, December, 1852. Accounts of two cases, with remarks, and references to the literature of the subject, by Prof. H. J. Bigelow, Dr. Morland, and Dr. John S. Flint, are contained in an article which has appeared, since the printing of this work has been commenced, in the Boston Med. and Surg. Journal, vol. lxi. No. 2, August 11, 1859.

[3] London Medical Times and Gazette, May 24, 1856, and the American Journal of Medical Sciences, July, 1856, page 258.

in the ordinary form of bronchocele or goitre. According to Dr. Graves, the limited extent of the enlargement is a point distinctive of its connection with an abnormal condition of the heart. Both lobes of the thyroid body may be affected equally, or the size of one lobe may be disproportionately increased. Having attained to a certain bulk, the enlargement ceases to be progressive, and the swelling remains, temporarily or permanently, in a stationary condition. Its size has been observed, however, to fluctuate between certain limits, varying with the action of the heart. A strong arterial pulsation is felt when the hand is placed over the tumor, and frequently a tremor or thrill resembling that communicated by an aneurismal varix. Dr. Stokes cites an instance in which it was mistaken for aneurism. An arterial bellows murmur is produced within the tumor, and, in some instances, also a continuous venous hum. The latter is sometimes musical, and has been known to be sufficiently loud to attract the attention of the patient. Unusual pulsation of the arteries of the neck coexists with arterial murmur. The inferior thyroid arteries have been found to become much enlarged. The cervical veins are notably dilated, and yield a continuous murmur or hum.

These phenomena pertaining to the thyroid body in the great majority of cases have been observed in females. They have not been observed to occur before puberty. Hysterical symptoms have been present in some instances. Neuralgia is an occasional concomitant. Anæmia coexists generally. As regards the heart, the cases have been characterized by long-continued, excessive action of the organ, or frequently recurring palpitation. The physical evidence of organic disease has in some instances been present and sometimes wanting. The amount of information obtained by examinations after death is, as yet, meagre. In a fatal case reported by Dr. Marsh with the autopsical appearances, the heart was enlarged, especially the auricles, and the auriculo-ventricular valves on both sides, more especially on the right side, were thickened by morbid deposit. In another instance, communicated to Dr. Stokes by Professor Smith, of Dublin, slight aortic lesions existed, and the left ventricle was largely hypertrophied and dilated. In a third case, detailed by Dr. Begbie, the heart was large, soft, and flaccid; all the cavities, but especially the ventricles, were dilated, and the valves were sound.

My own experience furnishes but little with regard to this subject. It is not improbable that examples may have fallen under

my observation without having been noted, inasmuch as my attention has been directed to the subject only for the last four or five years. Within this period I have met with three instances of an enlarged and pulsating thyroid body in connection with a notable degree of cardiac disorder. In all these instances the patients were females. In one the patient was twenty-seven years of age, had been married eleven years, and had had five children, four of which she had nursed for twelve months. At the time of my examination she was five months advanced in pregnancy, and her youngest child was two years of age. The enlargement of the thyroid body commenced shortly after the last confinement. It was considerably enlarged, and pulsated strongly. It is not noted whether murmurs and fremitus existed in the tumor. She had suffered from inordinate action of the heart for a year and a half. The action of the heart was not irregular, and the pulse numbered one hundred and twelve. Her appearance was not in a marked degree anæmic. She was affected with intercostal neuralgia. The impulse of the heart was abrupt and smart, with violent shock, but no heaving. No endocardial murmur was discovered. The sounds were normal. Owing to the large development of the mammæ, it was not easy to determine the size of the heart. The patient was from a distance, and the subsequent history of the case is unknown. In the second case the age of the patient was fifty-seven. She was the wife of a farmer, and had been accustomed to hard work in the management of a dairy. Menstruation had ceased three years before. Thirteen years prior to my examination she had acute articular rheumatism. For more than a year she had been troubled with palpitation, occurring especially at night, and produced by any excitement. It was unattended by dyspnœa. Considerable enlargement of the thyroid body had existed for about a year. On my first examination no murmur was discovered. The situation of the apex-beat was normal; the force of the impulse was not increased, and no abnormal modifications of the heart-sounds were observed. A year afterward a soft systolic bellows murmur over the apex was discovered, without any other signs of organic disease. The palpitations still continued. The subsequent history is not known. The third case came under observation quite recently. The patient is a young girl aged nineteen, presenting a healthy aspect, the countenance not denoting anæmia. She had been conscious of increased action of the heart for a year. The pulse was one hundred and twenty, and had been even more frequent. The enlargement was

moderate, the body on the right side being more affected than that on the left. There existed strong pulsation and a bellows murmur on both sides over the tumors; also arterial bellows murmur and venous hum over the carotids and jugular veins. The heart-sounds in this case were intensified, but otherwise not abnormal. No endocardial murmur was discovered, and the heart did not appear to be enlarged. Prominence of the eyeball existed in this case.

The view taken by Drs. Graves and Stokes of the pathological connection supposed to exist between enlargement of the thyroid body and an abnormal condition of the heart is, that the former may occur in consequence of undue rapidity, irregularity, and force of cardiac action, persisting for a sufficiently long period. Agreeably to this view, the heart may or may not be affected with organic disease. Prolonged functional excitement appears to be regarded as the essential abnormal condition, and this condition may be associated with different lesions, or the organ may be structurally sound. In the majority of the cases which have been reported, physical signs have been present denoting either enlargement or some organic change. It is evident that the subject claims farther clinical study, and that the data are at present insufficient to warrant positive conclusions respecting the nature and extent of the relations existing between the phenomena pertaining to the thyroid body and those referable to the heart.

Prominence of the eyes has been observed as a concomitant of the affection of the thyroid body. It was noted by Dr. Parry in one of the seven cases which he recorded of the latter affection. It coexisted and was more or less marked in the cases reported by the other observers whose names have been mentioned. The appearance of the eyes is peculiar and striking. The protuberance of the globes renders visible a broader portion of the tunica albuginea surrounding the cornea than usual, and gives to the countenance a wild, staring expression, which, as Dr. Taylor remarks, when once seen, will never be forgotten. The conjunctiva and other coats may remain clear and transparent, and sometimes the eyes acquire an unusual brilliancy. The pupil is not affected. Vision is unimpaired. The condition is unattended by pain. In some instances the projection of the eyeballs is so great that the lids are unable to cover them, and the patient sleeps with the eyes partially open; yet Dr. Stokes states that under these circumstances the sense of sight did not suffer, and ophthalmia was not developed in a case which remained under his observation for more than a year. The

affection is sometimes developed suddenly. In a case referred to by Dr. Stokes it became apparent after a long-continued fit of coughing and retching. An instance in which it occurred during a single night is related by Robert Taylor.

The rationale of this remarkable appearance of the eyes is not fully understood. Dr. Stokes attributes it to enlargement of the eyeballs from an increase in the vitreous and aqueous humors, and considers it therefore as denoting a form of hydrophthalmia, or general dropsy of the eye. This explanation is hardly consistent with the absence of pain, of defect of vision, etc. It is, moreover, disproved by a fact stated by Robert Taylor, viz., the balls can be readily replaced by gentle pressure. The hypothesis of Mr. Dalrymple is more plausible. He attributes it to "an absence of the proper tonicity of the muscles by which the eyes are retained in their natural positions in the orbit, and some amount of venous congestion of the tissues forming the cushion behind the globes."[1] From the cases which have been reported it would appear that it is almost invariably associated with enlargement of the thyroid body, and that the latter first occurs. Hence there is room for the conjecture that it is incidental to enlargement of the thyroid body, and if dependent on an abnormal condition of the heart, it is so indirectly. Another conjecture is, that enlargement of the thyroid body and prominence of the eyes are different effects of a common pathological condition, whatever it may be, the latter effect being less frequent, and rarely occurring without the former.

With regard to this subject, I can contribute from my own experience even less than with regard to the subject just considered. I have met with a single example only, and, by a curious coincidence, it has occurred since I commenced this chapter. The case has been already referred to, being the third of the three cases of which an account was given in connection with the subject of enlargement of the thyroid body. Abnormal rapidity of the heart's action preceded the prominence of the eyes for seven or eight months. The projection was noticed all at once, and the patient states that it followed violent fits of coughing. Both eyes were affected, but the right much more than the left. The thyroid body was also more enlarged on the right side than on the left. The general health of the patient has been, and is now apparently good. She is 19 years of age, and presents a healthy aspect. The countenance does not denote anæmia, but a loud venous hum, as well

[1] Quoted from article by Dr. Taylor.

as arterial murmur, exists in the neck. She had had for several weeks a dry irritable cough, without any other evidence of pulmonary disorder. The heart did not appear to be enlarged; the sounds were normal, except that they were intensified, and no endocardial murmur was discoverable. The pulse had ranged from 120 to 130. At the time of my examination, it was 120. At that time, the left eye projected but slightly, and the projection of the right eye had diminished. Vision was unaffected; the pupils were natural; pressure gave no pain, and there was no injection of the vessels. An instance was once related to me by a non-medical friend, which is worthy of being referred to, from the fact that the prominence of the eyes was attributed by the patient and her family to the injudicious use of iodine to resolve enlargement of the thyroid body. The medical attendant was held responsible for the occurrence, and I suspect was not prepared to vindicate himself by asserting the existence of a pathological connection between the two affections. The patient, as I have learned, had suffered much and long from disturbed action of the heart prior to the development of these affections. In the case which has recently come under my observation, a tonic course of treatment has been pursued and is still continued, consisting of preparations of iron, generous diet, and exercise in the open air.

It is evident that further investigation is needed to elucidate the pathological character and relations of this, as well as the associated affection previously considered. With our present knowledge, the agency of an abnormal condition of the heart in its production, directly or indirectly, cannot perhaps be considered as conclusively determined; and if it be referable to the heart's action, the nature and extent of its connection with the latter, remain to be ascertained. These points are of interest and importance, but their discussion here would be out of place. It is proper, however, to add that some writers attribute both affections to the co-existing anæmia which exists in a marked degree, in the majority of cases. This view is taken by Dr. Begbie,[1] and also by Dr. Taylor, of New York, and by Robert Taylor, of London, in the articles already referred to.

It is important, of course, to discriminate between prominence of the eyes, supposed to be incidental to an affection of the heart, and the enlargement or protrusion due to certain diseases of the eye-

[1] "Anæmia and its Consequences; Enlargement of the Thyroid Gland and Eyeballs; Anæmia and Goitre, are they related?" *Edinburgh Monthly Journal*, vol. ix. Feb'y 1849.

ball, tumor within the orbit, cerebral disease, etc. This is not difficult. The fact that both eyes are simultaneously affected; the coexistence of enlargement of the thyroid body, of disordered action of the heart, and, generally at least, of anæmia; the absence of pain, of local inflammation, of defective vision, and of symptoms pointing to the brain as the seat of disease, render the diagnosis sufficiently easy.

The prognosis in cases in which enlargement of the thyroid body and prominence of the eyes occur, exclusive of the cardiac lesions with which they may be associated, is not unfavorable. Danger to life is not involved in the occurrence of one or both of these events. Their liability to continue indefinitely, the inconvenience attending them, and the deformity which they occasion, are, however, not inconsiderable evils. The final result will depend on the coexisting morbid conditions. It would be injudicious to encourage confident expectations of complete recovery; but marked improvement, if not cure, may in many, if not most instances, be expected. The projection of the eyeballs often disappears, or diminishes so as to leave only a slightly unnatural expression. The thyroid body becomes, after a time, in favorable cases, solid and more or less lessened in size, the pulsation, thrill and murmurs gradually disappearing. These changes take place very slowly. The statements just made embody the conclusions drawn by Dr. Stokes from his own experience and that of others; but the accumulation of a larger number of cases is desirable to serve as the basis of our knowledge of the natural tendencies of the affections, and the extent to which they are amenable to treatment.

The indications for treatment relate mainly to the associated pathological conditions. Inordinate activity of the heart as regards rapidity or force, and irregularity of action, claim those measures which are suited to allay the morbid irritability of this organ. The consideration of this object of treatment belongs properly to the subject of functional disorders of the heart, to which a distinct chapter will be devoted. Coexisting anæmia calls for the treatment suited to that condition. Disorder of the catamenia (which is common), and, in fact, of any of the functions of the body, is to be remedied if practicable. It is yet to be ascertained whether any special treatment, aside from these indications, may be employed with advantage. Organic affections of the heart, if present, demand the treatment already considered.

REDUPLICATION OF THE HEART-SOUNDS.

Reduplication, or doubling of one or both of the sounds of the heart, is a rare auscultatory sign which was not considered in treating of organic affections, because it has not been found to be incidental to any particular form of cardiac lesion. Although, as a physical sign, it has not much practical value, it claims attention as denoting a curious and interesting aberration of the heart's action, and it is important for the practitioner to be prepared to recognize it, in order that he may appreciate what would otherwise be an unintelligible anomaly. Either of the sounds may be doubled singly, or the two sounds may be reduplicated. Reduplication of the second or diastolic sound is the variety oftenest observed. This is not extremely uncommon. Instances in which the first sound is doubled are much more infrequent; and examples of the reduplication of both sounds are so rare that they are justly included among the curiosities of clinical experience. Using for the present purpose the expression *tic-tac* as the symbol representing the normal sounds, the three varieties of reduplication may be verbally expressed thus: Doubling of the first or systolic sound by tic tic-tac; of the second or diastolic sound by tic-tac tac; of both sounds by tic tic-tac tac. In the two first varieties, or when one only of the sounds is doubled, three sounds occur during a single beat or revolution of the heart; in the last variety, or when both sounds are reduplicated, four sounds are heard with each revolution or beat. Bouillaud compares the rhythmical succession of the triple sounds, when the second sound is doubled, to the dactyle in poetical metre, and the rebound of a hammer on an anvil; and when the first sound is doubled, to the tattoo (rappel) of the drum, or the sounds of the feet of a galloping horse.

Bouillaud claims to have been the first to describe reduplicated heart-sounds. He states that, in the first edition of his treatise on *Diseases of the Heart*, published in 1834, he was able to cite but a single instance; but, before the appearance of the second edition in 1841, he had collected several examples, so that the phenomena could no longer be discredited, as they had been by some.[1] The

[1] Leçons Cliniques sur les Maladies du Cœur, etc., par M. Bouillaud, recueillies et rédigées par le Dr. V. Racle, Paris, 1853.

reality of the sign has been abundantly confirmed by other observers.

Of twelve cases of different varieties of reduplication, the histories of which are given in the second edition of the treatise by Bouillaud, in one only were both sounds doubled. In that instance the sounds were at first tripled, and afterwards became quadrupled. The four sounds for each beat were very distinct, and the correctness of the observation was verified by several observers sufficiently skilled in physical exploration. In this instance double pulsation of the arteries was also observed. The details of two cases are given by another French author, Dr. Charcelay, of Tours;[1] one of these cases came under his own observation, and the other is quoted from a thesis by M. Pressat, published in 1837. Reference will be made to these cases presently. An example as striking as any on record was reported by me in 1855.[2] The case came under my observation at the Louisville Marine Hospital in 1853. The patient was a sailor, aged twenty-seven, and was admitted for a cough which he attributed to taking cold six weeks before. On comparing the pulsations of the radial artery with the heart-sounds, there existed four sounds for each pulse. The number of double sounds was precisely twice as many as the number of radial pulsations per minute. This exact ratio was invariably preserved whenever the comparison was made for at least seventeen days. During this period, the comparison was made by several persons besides myself. The carotid pulse, however, occurred in the ratio of one to every two sounds of the heart; in other words, it was precisely twice as frequent as the radial pulse. The heart was evidently enlarged, and a feeble, short murmur was heard at the apex. The apex-beat was not distinctly appreciable either to the eye or touch. The patient was walking about, and was able to take active muscular exercise. Subsequently, the face and lower limbs became somewhat œdematous, and he suffered from dyspnœa. These symptoms disappeared, and he was discharged quite well, after being two months in hospital. When discharged, the pulse and two sounds of the heart were in normal ratio, being eighty-four per minute, and the bellows murmur had disappeared. The

[1] Mémoire sur plusieurs Cas remarkables de défaut de Synchronisme des Battements et des Bruits des Ventricules du Cœur. Archives Générales de Médecine, 1838.

[2] Buffalo Medical Journal, vol. ii., No. 1, May, 1855. Also Western Medical Journal, 1855.

patient remained apparently healthy, performing active labor for five years afterwards. He then died with some affection foreign to the heart, and a post-mortem examination with reference to the latter was made by my friend, Prof. T. G. Richardson, now of the University of Louisiana. The heart was moderately enlarged, without any appearances denoting valvular disease.

Reduplication of one of the heart-sounds, as already stated, is not extremely uncommon. In most cases it is the second or diastolic sound which is doubled. I have met with a few examples of this variety. It must be comparatively rare to meet with cases in which the first sound is alone doubled. None have, as yet, fallen under my observation.

Reduplications may be perceived only when the stethoscope is placed at certain points within the præcordia. If the instrument be moved to other points, the sounds, as regards number, are normal. Dr. Walshe gives the results of his observations with respect to the particular situations in which, in different cases, he has found the first and second sound, respectively, doubled, as follows: The first or systolic sound may be double at the apex over the left ventricle, single over the right ventricle, and either single or double at the base; it may be double over the right ventricle, and single over the left ventricle at the apex and at the base; it may be double at the base, single at the apex over the left ventricle, and imperfectly reduplicated over the right ventricle. The second, or diastolic sound may be double at the base and single at the apex; or it may be double over the right ventricle and single over the left ventricle at the apex and at the base. In the cases in which I have observed reduplication of the second sound, it has been noted as occurring at the base. In the instance in which both sounds were reduplicated, they were all distinctly heard by auscultating over the body of the heart. In the case reported by M. Pressat, the four sounds were heard within a space quite limited, corresponding to the interventricular and interauricular septa. In this situation, the sounds succeeded each other rapidly, without interruption, resembling, as the author states, the blows of a *batteur de plâtre.*

The cardiac lesions found after death in cases in which reduplications have been observed, are not uniform. In all of five cases reported by Bouillaud, valvular lesions existed, involving contraction at the aortic orifice in three, and at the mitral orifice in two cases. The heart was enlarged in all these cases. Tricuspid insufficiency, with hypertrophy, was found in the case of quadrupled

sounds reported by Dr. Charcelay, the other valves being sound. In two cases observed by Dr. Bellingham, in which the first or systolic sound was doubled, the right ventricle was much dilated and hypertrophied, and the valves at the left side of the heart were sound. The condition of the tricuspid and pulmonic valves is not mentioned. In the case reported by me of reduplication of both sounds, as already stated, the heart five years afterwards, when death occurred, was found to be enlarged without valvular disease. In another case under my observation in which reduplication of the second sound was ascertained a few days before death, the patient died of pericarditis and pleuritis developed in connection with Bright's disease. The heart, on examination after death, was greatly enlarged, but the valves were sound. Bouillaud was led by the post-mortem appearances in the cases which he had observed, to conclude that reduplication does not occur, except in connection with organic disease involving valvular lesions with either constriction or insufficiency. The facts just stated suffice to disprove this conclusion. Valvular lesions were wanting in the case observed by me in which both sounds were doubled. The absence of valvular lesions, moreover, is evidenced by the physical signs denoting their presence being deficient in a certain proportion of cases. Valvular lesions may or may not be present; they are not essential to the presence of this sign. In all the reported fatal cases which have fallen under my notice, however, as well as in the few cases that have come under my observation, the heart was more or less enlarged. It is probable that reduplication never occurs except in cases of enlargement of the heart.

The mechanism of reduplication of the heart-sounds is an interesting point of inquiry. The explanation at first offered by Bouillaud was, that the systolic and diastolic movements of the ventricles take place synchronously, but consist each of two distinct efforts (*reprises*); that is to say, each systole and diastole is divided into two acts, and each act attended by a sound. The disorders of rhythm, according to this hypothesis, is analogous to interrupted or jerking respiration, called by the French writers, *entrecoupée*. It is assumed in this explanation, that the diastolic sound of the heart is due to an active dilatation of the ventricles, and not to the recoil of the aorta and pulmonic artery, the latter, probably, being in fact the immediate cause of the expansion of the semilunar valves which occasions this sound. In his *Leçons Cliniques*, published in 1853, however, Bouillaud adopts the explanation now

generally received, as more satisfactory than that previously offered by himself. Agreeably to this explanation, the reduplication is caused by the failure of the two ventricles to contract in unison. This is the hypothesis advocated by Dr. Charcelay in the article already referred to. It has been adopted by late writers on diseases of the heart, so far as I know, without an exception. Dr. Charcelay, in the case of quadrupled sounds reported by him, regarded as proof of the non-synchronism of the ventricular contractions, the occurrence of jugular pulsations which were not synchronous with the pulsations of the carotids. Tricuspid insufficiency in that case was shown to exist by the examination after death, and it is highly probable that the alternate pulsations of the vein and the artery were due to the contractions of the two ventricles not being in unison. The proof, however, is not complete, since the jugular pulsations, which were not synchronous with those of the artery, may have been produced by contractions of the right auricle. In the example reported by me of reduplication of both sounds, the pulsations of the carotid artery occurred in the ratio of one to two of the heart-sounds, being precisely double the pulsations of the radial artery. Bouillaud also states that, in the example observed by him, there was reduplication of the arterial pulse. How is this to be reconciled with the hypothesis of non-synchronism of the contractions of the two ventricles? Unless accounted for, it certainly is inconsistent with that hypothesis. It may be accounted for on the supposition of a *bis feriens* or dicrotic pulse: that is, after an arterial diastole produced by the contraction of the left ventricle, the recoil of the aorta was sufficient to give rise to a second arterial diastole in the carotid, sufficiently strong to be perceptible here, but, in my case, not in the arteries more remote from the heart.[1] On the whole, with our present knowledge, the phenomena of reduplication are explained most satisfactorily, by supposing that the two ventricles fail to contract in unison. According to this rationale, the tricuspid and mitral valves are made tense, not simultaneously, but successively, in consequence of the contraction of one ventricle being completed before that of the other; and the semilunar valves of the aorta and pulmonic artery expand alternately, instead of coincidently, since, in consequence of the difference in time between the completion of the contractions of the

[1] A dicrotic pulsation of the carotids, when the movements of this artery are visible, is sometimes distinctly apparent to the eye.

right and left ventricle, the recoil of the coats of each of these two vessels does not occur at the same instant. Hence, the conditions for the doubling of both sounds always exist, although one sound only, either the first or second, may be reduplicated; but it is readily conceivable that one only may be reduplicated with intensity sufficient to be appreciable by auscultation. It is also intelligible that the reduplication is often perceived only within a limited portion of the præcordia.

Dr. Walshe remarks, that "the real interest of reduplications arises out of their bearing on the theory of the heart-sounds." He asks, "How is the fact that the second sound may be continuously doubled at the base, and perfectly pure and single at the apex, explicable on the simple sigmoid theory of the second sound?" He adds, "A double sound does not become single by conduction over so short a space." This fact seems to me to be susceptible of an easy explanation, without conflicting with the theory which refers the production of the second sound to the semilunar valves. Examinations of the healthy chest show that the pulmonic second sound is weak, as compared with the aortic second sound. The former is only distinguishable, as a rule, in the first, second, or third intercostal spaces on the left of the sternum. The second sound heard over the apex, and elsewhere more or less removed from the points just named, emanates from the aorta. Hence, when the second sound is reduplicated at the base of the heart, the reduplication may not extend to the apex, simply because the pulmonic sound is not propagated so far. Owing to the relative weakness of the pulmonic second sound, the reduplication may be appreciable at the base of the heart on the left, and not on the right side of the sternum. Again, the fact that "the first sound may be single at the left apex and at the base, while it is distinctly reduplicate at the right apex," appears to Dr. Walshe to denote that the first sound consists of a ventricular and an arterial portion, and that the two are separated on the right side of the heart. But this fact seems to be explicable readily by the weakness of the tricuspid valvular element of the first sound, as compared with the mitral valvular element. The tricuspid valvular element is appreciable only over the right ventricle; hence a doubled first sound may be heard in that situation only, simply because the tricuspid sound is not strong enough to be transmitted to the left apex and to the base. Finally, Dr. Walshe cites the fact that the second sound may be single at the base and double at the left apex, as tending to show

strongly the partial origin of the second sound within the ventricle. This fact is not so readily explained, and I cannot but think that whenever the second sound is doubled at the left apex, it will be found to be reduplicated over the pulmonic, although it may be single over the aortic artery. The pulmonic sound may, in some instances, be propagated as far as the apex of the heart, especially when the right ventricle is hypertrophied, while it is not appreciable over the aorta.[1]

Reduplicated heart-sounds are distinguished, in general, without difficulty, if the auscultator be prepared to recognize them by a proper knowledge of the subject. The occurrence of three sounds with a single beat is easily determined, and the rhythm shows, at once, whether it be the first or the second sound which is doubled. The latter, it is to be borne in mind, is reduplicated much oftener than the former, and this variety of reduplication, certainly as a rule, will be most marked, and perhaps perceived exclusively at the base of the heart. As just stated, it will be most likely to be discovered over the pulmonic artery, *i. e.*, in the second intercostal space on the left side of the sternum. Reduplication of the first sound is, I suspect, most likely to be observed over the right ventricle. The movements of the apex of the heart against the walls of the chest, in some cases, give rise to a prolonged and interrupted sound (element of impulsion) which may be mistaken for reduplication of the first sound. True reduplication of this sound is irrespective of the element of impulsion, being due to the disconnection of the tricuspid and valvular elements which enter into its composition. When both sounds are doubled, the sounds succeed each other so rapidly that the systolic and diastolic are hardly distinguishable by means of rhythm, duration, etc. In the case which came under my observation, there were over one hundred and sixty double sounds, or more than three hundred and twenty single sounds per minute! The fact of reduplication in such cases is to be determined by comparison of the number of sounds with the apex-beat, if perceptible, or with the radial pulse. It is true that the apex-beat and the pulse, in certain cases of cardiac disease, do not correspond with the heart-sounds when the latter are not reduplicated; but when,

[1] In connection with these remarks, the reader is referred to the analytical decomposition of the two sounds of the heart into 1st, an aortic and a pulmonic valvular element composing the second sound; 2d, and a tricuspid valvular element, a mitral valvular element, and an element of impulsion composing the first sound. *Vide* Chapter I., page 58.

as in the case reported by me, the pulse is found regularly to bear to the heart-sounds precisely the relation of one to four, reduplication is to be inferred. If neither the pulse nor apex-beats are sufficiently appreciable to be enumerated, reduplication of the two sounds can hardly be discriminated with certainty from excessive rapidity of the heart's action. This was the case for the first day or two in the instance which came under my observation. In this instance the reduplication was singularly regular and persisting. Generally it is transient, occurring at irregular intervals, and varying in duration. The sounds may be tripled at one time and quadrupled at another time, a fact readily explained by the difference in the intensity of the heart's action at different periods.

The limited pathological import of reduplications of the heart-sounds, and of their diagnostic significance, are sufficiently apparent from the facts which have been presented. They occur in cases of organic disease of the heart, and probably never unless organic disease be present. But they do not occur exclusively in connection with any particular lesions irrespective of enlargement. Their value in diagnosis is therefore small. As a physical sign Bouillaud calls it *un signe de luxe*, from its superfluousness in diagnosis. As an aberration of the heart's action, it does not appear to be followed by any serious consequences. It does not render the prognosis more unfavorable. In the case observed by me of reduplication of both sounds, the patient recovered in a few weeks from this form of disorder, and remained perfectly well, notwithstanding moderate hypertrophy, for several years, dying at length of a disease foreign to the heart. In the case reported by M. Pressat, the patient was sufficiently restored in a short time to leave the hospital. It must be confessed that, with our present knowledge of the subject of reduplications, the interest belonging to it, if the antithesis be allowable, is rather scientific than practical.

The treatment will have relation entirely to the nature and extent of the coexisting cardiac lesions, together with the associated symptoms, etc. In this point of view, the subject does not claim consideration.

CHAPTER VII.

INFLAMMATORY AFFECTIONS OF THE HEART.—PERICARDITIS.

Acute pericarditis—Anatomical characters—Division into three stages or periods—White spots on the heart—Pathological relations and causation of pericarditis—Connection with acute rheumatism, with albuminuria or Bright's disease, with endocarditis, etc.—Symptoms of acute pericarditis—Symptoms referable directly to the heart, to the circulation, to the respiratory system, to the digestive system, to the countenance, position, etc., to the nervous system—Notable disorder of the brain and spinal cord in connection with pericarditis—Physical signs of acute pericarditis—Signs furnished by percussion, by auscultation, palpation, inspection and mensuration—Summary of the physical signs of acute pericarditis—Diagnosis of acute pericarditis—Prognosis in acute pericarditis—Treatment of acute pericarditis—Bloodletting, mercurialization, sedatives, revulsives or counter-irritants, opium, stimulants and eliminatives—Treatment prior to liquid effusion, during the period of liquid effusion, and after absorption of liquid effusion—Treatment when complicated with notable disorder of the nervous system—Subacute and chronic pericarditis, with and without liquid effusion—Symptoms, physical signs, and treatment—Paracentesis of the pericardium—Pneumo-pericardium and pneumo-pericarditis—Pericardial adhesions—Effects upon the heart and circulation—Diagnosis.

Inflammation affecting the heart may be limited to one of the anatomical structures which compose this organ. The investing serous membrane may be alone inflamed, constituting the affection called *pericarditis*. When the membrane lining the cavities, or the endocardium, is the seat of inflammation, the affection is called *endocarditis*. Inflammation of the substance or muscular tissue of the organ is distinguished as *carditis* or *myocarditis*. Although these different inflammatory affections may exist, each independently of the others, they are often associated.

In a large proportion of the cases of pericarditis, endocarditis coexists; and myocarditis very rarely occurs save in connection with inflammation of the investing or lining membrane of the heart. The intrinsic importance of these affections renders their study highly important. They are seated in an organ entitled to be called, *par excellence*, a vital organ. They involve, not infrequently, great suffering and imminent danger to life. They derive importance

from their remote consequences. The organic affections which have been considered in previous chapters, originate, in the majority of instances, in cardiac inflammation. The study of this class of affections has been rendered highly interesting and important by the developments of modern researches as regards their pathological relations, especially to rheumatism and renal disease, and by the improvements in diagnosis arising from the successful application of physical methods of examination.

PERICARDITIS.

Inflammation affecting the investing membrane of the heart, or pericarditis, is less frequent in its occurrence than endocarditis, but is a more serious affection as regards immediate danger, and, perhaps, also in view of its remote effects. This membrane is analogous in structure to serous tissue in other situations; and pericarditis does not differ essentially from pleuritis or peritonitis. The points of difference pertaining to the symptomatic phenomena and the dangers peculiar to it, depend on the comparatively small size of the pericardial sac, the fact that the substance of the heart consists of muscular tissue, the function of the organ and its physiological relations. In treating of pericarditis, the morbid changes incident to the disease, or its anatomical characters, are to be first considered. Its pathological relations and causation will next be most conveniently noticed. The symptoms, physical signs, diagnosis, prognosis, and treatment, will severally receive distinct consideration. This, like other inflammatory affections, is presented in an acute and a subacute or chronic form.

I shall treat, in the first place, of acute pericarditis under the foregoing heads, treating separately of subacute or chronic pericarditis; and, finally, the subject of pericardial adhesions will claim some attention.

Anatomical Characters of Acute Pericarditis.

The morbid changes found after death in fatal cases of pericarditis, do not differ essentially from those which belong to the post-

mortem history of other serous inflammations. The appearances vary according to the stage of the disease at which death takes place. Death rarely occurs at the very commencement of the inflammatory action. In some instances in which the disease has proved rapidly fatal, the serous surface has been found more or less reddened, mainly from injection of the vessels situated in the subjacent areolar tissue. The redness is arborescent and in specks or patches, the latter giving to the surface a dotted or mottled aspect. Mere redness, however, and vascular injection are not reliable as the sole evidence of inflammation.

The latter may be due to various causes which impede the circulation in the heart shortly before, or at the time of death; and the former may be produced after death by extravasated serum colored with the hæmatin of the blood globules. On the other hand, the redness which belongs to inflammation, here, as in other situations, may have existed during life and disappeared after death. Opacity of the membrane, alteration in its consistence, or the presence of lymph, are essential to constitute proof positive that inflammation has existed. Abnormal dryness of the membrane has been supposed to be an effect of incipient inflammation, and, immediately succeeding this, a glutinous or sticky sensation, communicated to the finger when passed over the surface. The latter condition, from its resemblance to that of some fishes when they have been several hours out of water, has been called by French writers, *poissonneux*. These signs, however, as well as vascularity and redness in specks or patches, are not, in themselves, sufficient anatomical evidence of pericarditis. They derive their claim to be included among the anatomical characters of the disease, from their association with the *ante-mortem* history, and with other *post-mortem* appearances which are unequivocal in their significance. Acute inflammation speedily leads to the exudation of coagulable lymph. This exudation takes place sufficiently to give rise to the characteristic solid deposit, in most cases, probably, within a few hours from the commencement of the inflammatory attack. The deposit, at first, of a jelly-like consistence, adheres slightly to the membrane, forming a thin layer, either limited to the base of the organ and about the roots of the large vessels, or extending, more or less, over the pericardial surface. The heart, at this stage, covered with thin, soft lymph, presents an appearance which has been compared to hoar frost, or to a "layer of liquid gelatine spread upon the parts with a camel's hair pencil." The process of exudation goes on, and the uncoagulable

or serous portion forms a liquid which accumulates within the pericardial sac. If the disease do not prove fatal during this period, the liquid is gradually resorbed, and adhesion of the pericardial surfaces brought into apposition follows.

It suffices to divide the disease into three stages, the division being based on the series of morbid events just mentioned. The brief period during which the membrane is supposed to be dry, or when a glutinous exudation is appreciable by the touch and not by the eye, is by some reckoned as the first or dry stage. Practically, this division is, to say the least, superfluous. The first stage may be considered as extending to the time when the accumulation of liquid is sufficient to be determinable during life by symptoms and physical signs. The second stage will embrace the period during which an appreciable amount of liquid continues. The third stage comprises the duration of the disease after resorption of the liquid. These stages may be called, respectively, the stage of exudation, of liquid effusion, and of adhesion. These terms, however, are open to criticism, and a more simple mode is to speak of the disease as consisting of three periods, viz., before, during, and after liquid effusion, the latter expression being understood as applying to a quantity of effused liquid sufficient to distend, more or less, the pericardial sac.

If the disease end fatally during the first period, or before much accumulation of liquid takes place, the heart presents a coating of lymph, varying in different cases in its thickness and extent of diffusion. The deposit is more apt to be present and is more abundant at the base than over the other portions of the organ. It may be situated on both the visceral and parietal surfaces of the pericardium, or it may be limited to the former. It is very rarely, if ever, found exclusively on the parietal surface. The lymph is soft, slightly adherent, being very easily removed, and presents, in different cases, diversities of appearance which subsequently become more marked, and will be presently noticed. The membrane is more or less opaque, and may present the arborescent and dotted redness already mentioned. The latter, however, are often wanting after death. In some instances, when the deposit of lymph has been removed, the general aspect of the organ is not, in a marked degree, morbid; in some instances, the membrane covering the heart is studded with prominences resembling the enlarged papillary bodies on the tongue; in other words, it presents a mammillated aspect. The opacity is due to infiltration beneath the membrane;

and this infiltration loosens the attachment of the membrane, so that it is detached from the heart with greater facility than in the normal condition of the organ. The exudation within the sac, in some instances, consists almost entirely of coagulable lymph, and an amount of liquid sufficient to be determinable does not occur during the progress of the disease. The affection in these cases is analogous to dry pleurisy; and they have been called cases of dry pericarditis. It is, however, rare for inflammation diffused over the pericardial surfaces, in other words, general pericarditis, to run its course without giving rise to considerable liquid effusion. When this effect does not occur, the inflammation is generally partial, that is, limited to a circumscribed portion of the membrane. This remark holds good equally with respect to pleurisy.

The accumulation of liquid sufficiently to be manifested by signs and symptoms, takes place at a period from the commencement of the disease, varying in different cases. The quantity becomes sometimes large enough to occasion distension of the pericardial sac in twenty-four or thirty-six hours from the date of the attack. In the majority of cases, three or four days elapse before this occurs. As just stated, in some exceptional cases it does not take place during the whole career of the disease. The amount of effusion also varies greatly in different cases. From four to six ounces of liquid may be determinable in some cases; and the quantity which may accumulate beyond this amount ranges from a few additional ounces to as many as eight pounds. A case is reported by Corvisart, and also one by Dr. Swett,[1] in which the accumulation attained to the maximum just stated. The distension in these cases was enormous, exceeding several times the limit to which the healthy sac is capable of being dilated by forcible injection of a liquid after death. Experiments made by Dr. Sibson to determine the latter point showed that, in the adult male at fifty years of age, the injection of twenty-two ounces of liquid dilated the sac to its utmost capacity.[2] Very great accumulation belongs rather to chronic than acute pericarditis. The quantity in the latter rarely exceeds two or three pints. The effused liquid is more or less turbid. It is sometimes transparent at the surface, resembling clear serum, but muddy and thick at the bottom. The turbidity proceeds from the admixture of lymph, and detached

[1] Lectures on Diseases of the Chest.

[2] Bellingham, op. cit., Part I., p. 22.

portions of the latter in flocculi or shreds are found in greater or less quantity at the dependent portion of the sac. This turbidity and the presence of flakes of lymph distinguish inflammatory effusion from the transudation which constitutes simple dropsy in this situation or hydro-pericardium. Occasionally, the liquid effused in pericarditis is sanguinolent, the blood probably being derived from the rupture of vessels in newly-organized structure. Laennec considered this as a variety of pericarditis, which he called hemorrhagic. The admixture of lymph sometimes renders it puruloid; and in some very rare instances the liquid may be truly purulent. In these instances the disease bears to ordinary pericarditis the same relation as empyema to ordinary pleurisy.

In the majority of cases in which acute pericarditis proves fatal *per se*, death occurs during the period of liquid effusion. On examination post-mortem, the pericardial sac is found to contain a certain quantity of turbid, puruloid, sanguinolent, or purulent liquid. The free surface of the sac, especially upon the heart, presents, in different cases, diversities of appearance, due to the quantity and disposition of the exuded lymph. The deposit is more abundant than it was prior to the liquid effusion. It has become more dense and more firmly adherent to the membrane. It is frequently laminated. Extending, in some cases, over the whole heart, or confined to certain portions, it forms a covering of variable thickness, which often has a reticulated appearance resembling gauze or lace-work; or, quoting comparisons by different observers, the appearance is frequently not unlike that of the section of a sponge, the interior of the gall-bladder, the second stomach of the calf, or a congeries of small earth worms. In other instances the lymph is rolled into ridges, giving to the surface of the heart a furrowed or wrinkled aspect. These ridges are so disposed in some cases as to give rise to an appearance which has been compared, poetically, by Hope to the undulations of sand on the sea-shore. Another disposition of the lymph is in the form of villous projections, giving to the exterior of the heart a shaggy appearance. In a specimen which I have, the entire surface of the organ is covered with closely set, fine filaments from two to four lines in length. This peculiar appearance probably is that to which was applied formerly the name *cor villosum*, or hairy heart. Another variety still is the deposit of lymph in minute patches, thickly disseminated over the heart's surface. The diversities of appearance are explained by the movements of the visceral and

parietal surfaces of the membrane upon each other, caused by the elongation and rotation of the heart during its systole. They may be rudely imitated, as Laennec remarks, by rubbing together and alternately bringing into contact and separating two marble slabs, their surfaces having been covered with a layer of soft butter. When the pericardial sac is distended with liquid, the two surfaces may come together at certain points during the systole, and recede during the diastole. It is evident that this cannot occur when the pericardial sac is empty; the two surfaces must then be constantly in contact, friction of the surfaces only taking place, without separation. The deposit of lymph, as already stated, may be general or limited to certain situations. It is most prone to accumulate near the base and about the large arteries at their origin. It may be found exclusively or most abundant on either the anterior or posterior surface of the organ. It is rarely very abundant on the parietal surface of the membrane.

When death does not occur during the period of the continuance of an abundant liquid effusion, the latter is removed, sometimes quite rapidly, and in other cases slowly, by absorption, and the pericardial surfaces again come into apposition. In examinations after death, at a period somewhat remote from this occurrence, these surfaces are found united. Adhesion has taken place. This may be either mechanical or by means of organization. In some specimens the parietal portion, together with the fibrous sac, is simply agglutinated to the heart with a certain force, and, when detached, successive layers of condensed lymph are found to intervene between this portion and the exterior of the organ. These layers may sometimes be peeled off, one after the other, presenting the appearance of distinct, firm membranes. Those nearest the heart are sometimes reddened with hæmatin. Collectively, they form a mass which may be nearly or quite an inch in thickness. This mode of adhesion is purely mechanical. These layers of lymph never take on organization. They are correctly called false membranes. It is doubtful if, under these circumstances, the organized adhesion ever occurs. The quantity of fibrin prevents this result. But in other instances, in which the intervening deposit of lymph is less abundant and dense, newly-organized structures are formed, and adhesion by a vital union takes place. The pericardial sac may be in this way completely obliterated. It was probably cases of this kind which, coming under the observation of some of the old anatomists, led them to conclude that the pericardium was

sometimes wanting. Union by an organized attachment may be partial. Sometimes it is limited to certain points, and, the newly organized structure becoming elongated by the movements of the heart, the opposing surfaces are connected by membranous bridles or bands. The pericardial surfaces may become firmly adherent over one side of the heart, and the remainder of the sac contain a considerable quantity of liquid. Two examples of this kind have fallen under my observation. In one, adhesion had occurred over the left half of the organ, and the quantity of liquid contained in the right half of the sac was so great as to extend far beyond the right margin of the sternum, giving rise to physical signs which were supposed to denote effusion into the right pleural cavity. In the other, the adhesion was over the right side, and the accumulation of liquid in the left half of the pericardial sac gave rise to a tumor which projected far beyond the left border of the heart. The process of adhesion may be completed within a period varying from a few days to several weeks. The strength of the adhesion is a test of its age. When recent, the attachment is easily broken, but it becomes extremely firm after the lapse of considerable time. The metamorphoses which take place in the deposit of lymph in certain cases, belong more appropriately to the anatomical history of chronic pericarditis, and will be noticed in that connection.

In examinations after death of bodies dead with various diseases, the symptoms during life not having pointed to the existence of any cardiac affection, one or more opaque patches are often found on the heart, generally situated on the anterior portion of the right ventricle, near the middle or toward the base of the organ. These are the white or milk spots (*maculæ albidæ vel lacteæ*) which have given rise to considerable discussion among pathologists. They vary in size from that of a half dime to a quarter of a dollar. Their form is variable, being round, oval, or irregular in their contour, and sometimes linear. The question is, To what extent are they to be considered as evidence of ancient pericarditis? They consist, in nearly all the instances in which I have examined with reference to this point, of lymph deposited in a thin layer, closely adhering, but which may be stripped off, leaving the surface of the heart beneath normal, except that it has lost somewhat of its naturally smooth and polished appearance. It is stated, however, that in some instances they proceed from opacity and thickening of the membrane itself. That they result from inflammation when they are due to a deposit of lymph, must be admitted. They constitute

evidence of partial or circumscribed pericarditis, but so limited in extent as not to give rise to any symptoms of disease or any evil consequences. Practically, they are of no importance. They are undoubtedly due to some local cause; and the most probable explanation is that given by Dr. Hodgkin and others, which attributes them to the attrition between the anterior surface of the heart and the thoracic walls. When the membrane itself is thickened, they are probably a kind of callosity caused by pressure. They are rarely found in subjects under the age of puberty, but very frequently after forty; and they are much more common in males than in females.

Pathological Relations and Causation of Pericarditis.

Acute pericarditis, as an idiopathic or primary disease, is extremely rare. The chances of its development, irrespective of any other disease, in a healthy person, are very few. In this respect it may be classed with acute gastritis and meningitis in the adult. In the vast majority of cases it is a secondary affection. It is developed either as a complication of some other affection, or in connection with some one of the diseases called general, or of the cachexiæ. It is understood, of course, that this remark does not apply to traumatic cases. The pathological relations of pericarditis, therefore, in this point of view, form an important as well as interesting portion of the etiological history of the disease. The affections of which it is an occasional concomitant are numerous, but in much the larger proportion of instances it occurs in the course of either articular rheumatism or renal affections involving albuminuria.

The attention of clinical observers has been directed to the occurrence of pericarditis in cases of rheumatism only within the last thirty years. The occasional association of these affections was noticed by Pitcairn in 1788, Dundas in 1809, and by Dr. Wells in 1812; but the existence of a pathological relation between them was not fully recognized prior to the publications of Dr. Latham and Dr. Elliotson in 1829. The subsequent observations of Bouillaud, Hope, and others, established the fact that, in a large proportion of the cases of pericarditis, the disease occurs in connection with rheumatism. Although it is observed much less frequently than endocarditis in this connection, the proportion of the cases of

acute rheumatism in which it becomes developed is tolerably large. Of 847 cases collected from various sources and analyzed by Dr. Fuller, it existed in 142, being in a ratio of about 1 to every 6 cases. These cases were reported by six trustworthy observers, and it is worthy of note that each collection of cases gives not far from the same ratio as when they are analyzed collectively.[1] Of 19 cases of recent pericarditis, the histories of which, recorded by myself, are before me, in 6 the affection occurred manifestly in connection with rheumatism. Statistics show that rheumatism is more likely to become complicated with pericarditis in proportion to the youth of the patients affected. Thus, Dr. Fuller deduces from cases reported by different observers that it occurs in a ratio of more than one-third under the age of 15; of a little less than one-fifth between the ages of 15 and 20; of less than one-tenth between 20 and 25; whilst above the age of 25 the ratio diminishes with even greater rapidity. A decided influence, therefore, in the production of the disease, in this connection, pertains to age. It appears also to occur in a larger ratio in females than in males.[2] The liability to pericarditis appears to be greater, other things being equal, in proportion to the acuteness and severity of the rheumatic attack, as denoted by the intensity of the local symptoms and the febrile movement. It is rarely developed in the subacute form of the latter affection; the chronic form, and the affection called muscular rheumatism, do not involve a liability of its occurrence, and the same may be said of gout. It has been observed to become developed oftener in the first than in subsequent attacks of rheumatism. It may occur at any period during the course of the rheumatic affection, but in the majority of cases it is developed between the fourth and twelfth days. Several instances have been reported in which it preceded the affection of the joints; I have met with one instance of this kind, and in one case which came under my observation it occurred coincidently with an affection of the wrist. According to Dr. West, it oftener takes precedence of the affection of the joints, and is more apt to occur coincidently with, or shortly after, the latter, in children than in adults.[3]

What is the nature of the pathological relation existing between pericarditis and acute rheumatism? After the fact of a relation was

[1] On Rheumatism, Rheumatic Gout, and Sciatica. By Henry William Fuller, M. D., etc. Am. edition, 1854, p. 216.

[2] Fuller, op. cit.

[3] On Diseases of Children, second American edition, p. 304.

ascertained, it was supposed that a transference of the disease from the joints to the heart took place, and that the pericarditis was due to change of seat, or metastasis. This view is disproved by the fact that the inflammation of the pericardium does not involve necessarily, nor indeed, generally, diminution of the articular inflammation. The reverse of this obtains in the majority of cases. Moreover, as has just been stated, the pericarditis in some instances precedes the affection of the joints. The same internal morbid condition which determines the latter, gives rise to the former. The one, as well as the other, is the local expression of a general or constitutional affection, involving, probably, blood-changes, in which consists the essential pathology of rheumatism. The pericarditis and the affection of the joints, in other words, are, alike, effects of a common pathological condition.

To inquire respecting the nature of the blood-changes which constitute this condition, does not fall within the scope of this work. As the affection of the joints in rheumatism exists in the larger proportion of cases without the development of pericarditis, so it is probable that rheumatic pericarditis may sometimes occur without the former. The liability of the pericardial membrane to become the seat of inflammation in rheumatism, is to be explained by the analogy of structure between this membrane and the tissues entering into the composition of the articulations.

Since our knowledge of the connection of albuminuria with certain affections of the kidney dates from the researches of Bright, published in 1827, it follows that the pathological relation existing between pericarditis and these affections has but recently been ascertained. Clinical observation within late years has abundantly established the existence of such a relation. Of 35 cases of pericarditis analyzed, with respect to causation, by Dr. John Taylor, Bright's disease existed in 13, and the development of the pericardial inflammation could not otherwise be accounted for.[1] Of 19 cases taken from my records, Bright's disease, or marked albuminuria, was present without any other apparent causative agency, in 3, and probably, also, in 2 additional cases. On the other hand, of 50 patients who had either died of Bright's disease, or who were ascertained to have this disease in an advanced form, acute pericarditis was found by Dr. Taylor in 5, or in the ratio of 1 to 10. Of

[1] On Some of the Causes of Pericarditis. Medico-Chirurgical Transactions, vol. xxviii. p. 453.

135 fatal cases of pericarditis analyzed by Dr. T. K. Chambers, the kidneys were diseased in 36.[1] In a collection of cases of pericarditis, including cases which recover as well as those which prove fatal, the disease is found to be developed much oftener in connection with acute rheumatism than with an affection of the kidneys. But in a collection of fatal cases of pericarditis, renal disease is found to coexist much oftener than acute rheumatism. The explanation of this is, pericarditis developed in connection with Bright's disease especially at an advanced stage of this disease, almost invariably ends fatally; whilst in connection with acute rheumatism, recovery takes place in a large proportion of instances.

What is the nature of the pathological relation existing between pericarditis and the renal affections generally included under the name of Bright's disease? Clinical observation shows that, in connection with the latter, serous inflammations are apt to become developed. The production of these inflammations, as well as other effects, are attributed to the accumulation of urinary principles in the blood, in consequence of the impaired excretory function of the kidneys. The intermediate morbid condition determining the pericarditis, is thus supposed to be uræmia. The urea in excess, or the products of its decomposition in the blood, act as poisonous agents, giving rise to inflammation of the pericardial and other serous membranes, among various pathological consequences. This is the explanation most consistent with our present knowledge.

Pericarditis is frequently associated with either pleuritis or pleuro-pneumonia. It has been inferred from this association that the inflammation extends from the pulmonary organs to the heart, in consequence of the proximity of the latter to the former, an inference which appears to be strengthened by the fact that the coexisting inflammation of the pulmonary structures is situated on the left, oftener than on the right side. But pleuritis and pleuro-pneumonia are diseases of frequent occurrence, and in the great majority of cases the inflammation does not extend to the pericardium. On the other hand, in cases of pericarditis developed in connection with acute rheumatism, the inflammation very rarely extends to the adjoining pulmonary structures. It is chiefly in non-rheumatic cases of pericarditis that this disease is associated with pulmonary inflammation. It is, therefore, more rational to conclude that between the latter and the former there exists no

[1] Decennium Pathologicum.

relation of causation, but that both are equally dependent on some internal, determining pathological condition. This condition belongs, in a certain proportion of cases, to disease of the kidney, and, in other cases, to some of the diseases occasionally giving rise to pericarditis, which are presently to be noticed. Although it is true that when pleuritis is associated with pericarditis the left pleura is oftener affected than the right, yet the right pleura is not infrequently the seat of the inflammation. Of seven fatal cases, of which I have preserved notes, the right side was affected in three. The pleuritis was double in three of these cases.[1] To show the relative frequency of the occurrence of pleurisy and pneumonia in cases of non-rheumatic as compared with rheumatic pericarditis, the following statistics by Dr. Taylor may be cited: In 24 cases of non-rheumatic pericarditis, pneumonia occurred in 12, while it occurred in only 4 of 16 cases of rheumatic pericarditis. Pleurisy occurred in 10 of 16 cases of the former, and in only 7 of 24 cases of the latter.

Pyæmia appears to rank next to Bright's disease as regards the frequency of its coexistence with pericarditis in fatal cases of the latter. It existed in 18 of the 135 subjects examined by Dr. Chambers, a ratio equal to that of rheumatism in this collection of cases. Pyæmia, at the same time, is likely to give rise to inflammation affecting serous structures in other situations. It is probably through the intervention of this blood affection that wounds of parts remote from the heart and surgical operations sometimes give rise to pericarditis. Of the nature of the pathological relation existing between pyæmia and pericarditis all that can be said with our present knowledge is, that the blood is so altered as to determine, among other results, inflammation of the pericardium.

The eruptive and continued fevers occasionally become complicated with pericarditis. It must be extremely rare for this complication to occur in connection with either typhoid or typhus fever; but examples of its development in the course of scarlatina and small-pox are not so uncommon. When it occurs as a sequel of scarlatina, of which I have met with an instance, it is probably dependent on the morbid condition incident to albuminuria. In the instance just alluded to, it was preceded by general dropsy and albuminous urine. The pathological relation with the essential

[1] Louis' statistics show coexisting pleuritic or pneumonic inflammation to be limited to the right side in one-third of the cases in which these pulmonary affections are associated with pericarditis.

fevers, also, involves certain internal causes pertaining probably to blood-changes.

Pericarditis has been observed to occur in cases of scorbutus. Its occurrence in this pathological connection was observed some years since during the prevalence of scurvy among sailors at St. Petersburg by M. Seidlitz, of that city; and a variety of the disease, said to be frequent on the extreme northern coasts of Europe, where scurvy is endemic, has been described by M. Kyber under the name of *pericarditis scorbutica*. Another observer, M. Karawagan, found that of sixty subjects dead with scurvy, thirty were affected with pericarditis. As described by these three writers, the pericarditis occurring in scurvy differs from the ordinary form of the disease, in the bloody character of the liquid effusion contained in the pericardial sac. It is, in fact, a species of hemorrhagic pericarditis.[1]

Purpura and cyanosis are other affections, characterized by a morbid condition of the blood, in connection with which pericarditis has been observed to become developed. It is, however, doubtful if the number of instances in which the association occurs is sufficient to show the existence of any special pathological relation.

Erysipelas and influenza are affections in connection with which pericarditis has been observed sometimes to become developed.

Tuberculosis of the lungs is sometimes associated with pericarditis. Of eleven fatal cases in which the pericarditis was recent, pulmonary tuberculosis coexisted in three. This association is probably due merely to coincidence. The tuberculous cachexia does not appear to give rise to inflammation of the pericardium, except in the very rare instances in which tubercle is deposited upon this membrane. The same remark is applicable to the carcinomatous cachexia.

As thus far considered, the causation of pericarditis has, for the most part, involved internal morbid conditions pertaining to certain general diseases, cachexiæ and blood-changes. In the great majority of cases, the disease is developed secondarily in some of these pathological relations, and, as already stated, more especially in connection with either acute rheumatism or renal affections accompanied by albuminuria. But various local causes may give rise to the disease. It may be produced by wounds penetrating

[1] Bellingham, op. cit., Part II. p. 263.

the pericardial sac, and by other injuries of the chest. In these cases the disease is traumatic. Abscesses formed in the liver occasionally open into the pericardial sac and occasion acute inflammation. Collections of softened tubercle in the lungs have been known to take this direction. Mediastinal abscesses may pursue the same course. The pericardium sometimes appears to take on inflammation in consequence of the local irritation excited by aneurismal tumors of the aorta. Enlargement of the heart probably, in some instances, leads to this result. The deposit of either tubercle or carcinomatous matter upon this membrane, happily extremely rare, is another local cause to which allusion has already been made.

Exclusive of the cases in which pericarditis is developed as a secondary disease, *i. e.*, dependent on some antecedent morbid condition, it may possibly occur as a primary or idiopathic affection. Instances, however, are so extremely rare that clinical observers of large experience declare they have never met with an example. On this point, Dr. Walshe remarks that "alleged idiopathic pericarditis becomes rarer every year, in proportion as the evolution of diathetic diseases grows more fully understood;" and he adds that he has never seen a positive case of the kind.

The influence of youth in the causation of rheumatic pericarditis has been referred to. Statistics show that non-rheumatic cases embrace a larger proportion of persons beyond the middle period of life. The average age in twenty-four cases of the latter, reported by Dr. Ormerod, was forty-two; while in sixty-one cases of the former, the average age was twenty-one. This disparity is intelligible when it is considered that, exclusive of acute rheumatism, pericarditis, in the majority of cases, is dependent on Bright's disease, and persons in middle life, more than the young, are liable to the latter. But no period of life is positively exempt from the liability to pericarditis. Numerous cases have been reported in which the disease occurred in infants but a few months old. In some of these cases the most prominent symptom was screaming of the infant. I met with a case, several years since, of the disease in a child eight months old, in which this was the chief symptom, the sudden, sharp, brief cry resembling that which is characteristic of meningitis, and leading to a suspicion of the existence of the latter affection.

In a very large proportion of cases of pericarditis, endocarditis coexists. In rheumatic pericarditis this is a rule to which there

are few, if any, exceptions. The rule does not hold good, at least to the same extent, in non-rheumatic pericarditis. The frequent association of the two affections will serve, in a measure, to account for the fact that when, on examination after death, the evidences of ancient pericarditis are discovered, the heart is often, if not generally, more or less enlarged, and valvular lesions are, at the same time, found. The remote effects of pericarditis on the heart, is a point of interest and importance, which will be considered in connection with the subject of pericardial adhesions.

Symptoms of Acute Pericarditis.

The symptoms of acute pericarditis vary according to the intensity of the inflammation, the amount of liquid effusion, and other circumstances, the effects of which are sufficiently manifest. But, irrespective of these, variations in different cases are observed, which cannot be traced to obvious differences in morbid conditions pertaining to the organ affected. In this respect, however, the disease does not differ from other inflammations, especially those affecting serous structures. The same is true of pleuritis, meningitis, and peritonitis. Certain of the symptomatic phenomena, such as pain, febrile movement, etc., are present in a marked degree in some cases, in a moderate or slight degree in other cases, and are sometimes wanting, when the appearances after death denote equal intensity and extent of the inflammation. There is, in short, often an apparent want of correspondence between the manifestations of the disease during life, and the changes ascertained after death, showing that the symptomatic events which belong to the clinical history of the disease, are influenced, in no small measure, by circumstances pertaining to other parts of the body than the organ affected, or to the general system. These circumstances are but little understood; but this remark is not more applicable to pericarditis than to various other local affections. This frequent want of harmony (if this term may be allowed) between local morbid conditions and symptomatic phenomena, is important to be considered in connection with diagnosis, prognosis, and treatment. It is this which invests the physical signs of disease, whenever they are available, with much of their great practical value. Pericarditis being associated, in the great majority of cases, with other affections, its own manifestations are, to a greater or less extent,

intermingled with, and obscured by those of the latter. This fact, together with the variations just alluded to, impairs considerably the general application of a descriptive history based on the clinical study of the comparatively rare cases in which the disease is isolated, and its symptomatic phenomena strongly marked.

The division of the career of the disease into three periods, based on facts pertaining to its morbid anatomy, is to be borne in mind. The symptoms undergo important modifications when an abundant accumulation of liquid takes place in the pericardial sac, in other words, during the second period; and, again, after absorption of the liquid, or during the third period.

In treating of the symptomatology of pericarditis, the symptoms which relate directly to the heart will be first noticed, and, afterward, those referable to different anatomical systems—the circulatory, respiratory, nervous, etc.

Symptoms referable directly to the heart.

The symptoms which relate directly to the heart, are pain, palpitation and tenderness.

Pain referred to the præcordia, is a prominent symptom in some cases. The character of the pain is burning or lancinating, and it is accompanied often by a sense of constriction. It is aggravated by inspiration; and the inspiratory acts are sometimes shortened in consequence, the number of respirations per minute being correspondingly multiplied. The pain is also increased by movements of the body. It may be referred to the region of the heart or to the epigastrium. In two cases under my observation, it was at first seated on the right side of the sternum, and shortly shifted to the præcordia. It may extend to the back, to the left shoulder, and down the left upper extremity, bearing some resemblance, if severe, to the pain in angina pectoris. The character of the pain, and the associated circumstances being the same in some cases as in pleurisy, the affection is liable to be mistaken for the latter. This not infrequently happens. Pleurisy, as has been seen, is, in many cases, associated with pericarditis, and the pain belonging to each affection separately, cannot, under these circumstances, be readily disconnected. The pain, also, is not unlike that incident to pleurodynia and intercostal neuralgia, and these affections may coexist with pericarditis. But pain is by no means invariably a prominent symptom of pericarditis. In the majority of cases it is either mode-

rate or slight in degree. It may be wanting. In a case recently observed in which pericarditis was consecutive to pleurisy of the left side, with large effusion, the heart being removed to the right of the sternum, no pain was experienced in the organ at the time of the attack, or subsequently. In another instance, the pain was slight, and referred to the region of the dorsal vertebræ. In most of the cases which have fallen under my observation, the pain has been either slight or moderate. I have never witnessed excruciating suffering from pain in connection with pericarditis, but instances have been reported. This symptom, thus, is variable in degree; it is not reliable as a constant symptom, and, when more or less marked, is not distinctive.

Upon what does the occurrence of pain depend? Bouillaud attributes it, in all cases, to coexisting pleuritis. But it may undoubtedly be present, and prominent as a symptom, when pleurisy does not coexist. It must emanate from the nerves of the heart; and, although it is difficult to explain the differences in the amount of pain in different cases which appear to be similar as regards the intensity of the inflammation, and to account for the absence of pain in certain instances, it is to be considered that the same difficulty is met with in other serous inflammations, for example, peritonitis. The increase of pain during the act of inspiration is explained by the friction of the pericardial surfaces in consequence of the depression of the diaphragm, and by the pressure upon the heart, of the lungs in the process of inflation. When pain is present, it belongs especially to the commencement of the attack and the early part of the disease. It diminishes or disappears when the inflamed surfaces become covered with lymph, and are separated by liquid effusion. At this stage it may give place to a sense of uneasiness or undefined distress referable to the præcordia, not amounting to positive pain.

Tenderness on pressure is a symptom frequently but not constantly present. Like pain, when present, it is variable in degree, It is rarely very marked. As pointed out by Hope, tenderness may sometimes be discovered on pressing upon the epigastrium beneath the cartilages of the ribs in a direction toward the heart, when it is not apparent in the præcordia directly over the heart. In order to constitute a symptom of pericarditis, it must be limited to the region of the heart. It is needless to say that when pleurisy, affecting the left side, coexists, the tenderness will be diffused, more or less, over the whole of that side. In acute rheumatism, pleuro-

dynia may occur, either with or without pericarditis, and diffused tenderness will then be present. In rheumatic pericarditis, I have found the whole of the left side exquisitely sensitive to pressure, without the physical evidence of pleuritis. As an isolated symptom, circumscribed tenderness is of little value, but, taken in connection with other symptoms, it is of importance. It is to be borne in mind that the absence of tenderness is not positive evidence against the existence of pericarditis.

Tenderness is doubtless due to an abnormal sensibility developed in the inflamed membrane. Why this sensibility is present in some cases and not in others, cannot be explained; but the same is true of other serous inflammations. In peritonitis, for example, the tenderness is generally great; but in a well-marked case at this moment under observation, this symptom is wanting.

Increased action of the heart is an effect of pericardial inflammation during the early part of the disease. The contractions are violent and sometimes irregular. The patient is conscious of an unnatural beating of the organ. This constitutes palpitation. Like the other symptoms referable to the heart, this is by no means constant, and it possesses, in itself, but little value inasmuch as it occurs in connection with the different forms of organic disease, and, also, as a purely functional disorder. Its importance depends on its association with other symptoms. It is sometimes strongly marked. The commencement of the disease may be characterized by tumultuous action of the heart. In cases of acute rheumatism, this should excite strong suspicion of pericarditis, and lead to a careful examination for more positive evidence of the disease. Palpitation belongs to the first stage of pericarditis. It is incompatible with much liquid effusion, and even if the latter does not occur, a secondary effect of the inflammation on the muscular substance of the heart, is diminished power of contraction, or incomplete paralysis. A similar effect is observed in cases of peritonitis, as shown by enlargement of the intestines, from the pressure of their gaseous contents.

Symptoms referable to the circulation.

The pulse, considered alone, in this, as in most diseases, does not furnish characters which, in a diagnostic point of view, are highly distinctive; but, the diagnosis being made, it gives important information respecting the condition of the heart. At the onset of

the disease, it corresponds to the increased muscular action of the organ, and is frequently strong, quick, vibratory, as well as more or less frequent and sometimes irregular. In proportion as the heart is weakened, in the progress of the disease, it becomes enfeebled; and when in conjunction with a certain amount of paralysis, the movements of the organ are mechanically restrained by the pressure of liquid effusion, the pulse is notably small and weak, with more marked disturbance of its rhythm. It represents, thus, the effects of the disease, vital and mechanical, on the circulation. Dr. Stokes thinks that the effect upon the heart's movements produced by the pressure of liquid in the pericardial sac is overrated, and he cites, in support of this opinion, the comparatively small disturbance of the circulation caused by dislocation of the heart in cases of empyema. But the condition of the organ in the two cases is by no means the same. When the heart is removed to the right of the sternum by the pressure of a large accumulation of liquid in the left pleural sac, the freedom of its movements is but little restrained in comparison with the effect of an abundant effusion within the pericardial sac. When the latter is distended to double or treble its normal capacity, it is truly surprising that the cavities receive sufficient blood for the circulation to be carried on. The fact that the circulation is not arrested, shows the force with which the blood is returned to the heart. In dwelling upon the atony or paralysis arising from the proximity of the inflamed membrane to the muscular tissue, Dr. Stokes seems to me to undervalue the mechanical effect of the presence of liquid. The latter effect is proportionate, other things being equal, not so much to the amount of liquid effusion as to the rapidity with which it takes place. If it accumulate slowly, the dilatation of the sac goes on *pari passu*, and the heart, speaking metaphorically, becomes accustomed to the pressure. If the quantity, on the other hand, become rapidly large, the sac does not readily yield, and the heart suffers from the compression in a more marked degree. Clinical observation sustains the correctness of these remarks. The pulse may be greatly enfeebled from the weakness of the heart induced as a secondary result of the inflammation, irrespective of effusion. But the pressure of liquid, especially when rapidly effused, affects the pulse to a still greater extent. A weak and small pulse belongs to the stage of effusion, and may be considered as representing, in a great measure, the extent to which the heart is mechanically restrained. As regards frequency of the pulse, Dr. Walshe remarks that it "is

subject to more sudden variations from the influence of emotional excitement and effort than in any other disease, perhaps." He adds that he has known a very gentle movement of the trunk raise the pulse from 80 or 90 to 130 or 140. It is often found to vary notably on different days, without any obvious cause.

During the progress of pericarditis, then, as a rule, the pulse is at first more or less increased in frequency, and also in force and quickness; and, afterwards, from the combined effects of diminished muscular power and the pressure of liquid effusion, it is irregular, weak and small, the frequency being generally still more increased. But to this rule there are exceptions. The frequency during the first stage is, in some cases, not greater than in health. Dr. Graves, indeed, states that he has observed it to be less frequent than in health. It may continue regular during this stage, and present no marked deviation from its normal characters. During the second stage, the effect of even a large accumulation of liquid is sometimes not very marked. It may retain considerable force and volume when the extinction of præcordial impulse and other physical signs show distension of the pericardial sac.

In cases of rheumatic pericarditis, the pulse is more or less accelerated prior to the development of the heart affection. The influence of the latter, therefore, cannot be estimated with precision. A sudden change in frequency, or other characters, during the course of acute rheumatism, occurring when no joints are newly attacked, and irrespective of any obvious cause, should lead the practitioner always to direct his attention to the symptoms and signs of cardiac disease.

The obstruction to the circulation incident to prolonged accumulation of liquid in the sac may be sufficient to give rise to œdema of the lower extremities and face. In general, however, œdema involves coexisting disease of kidney, or organic lesions of the heart.

Lividity of the lips, face, etc., may be due alone to the pressure of liquid and weakness of the heart. As a symptom of pericarditis, exclusive of other affections, it belongs to the second stage of the disease, and denotes an alarming degree of obstruction. The pulse will be found to be, at the same time, extremely feeble and irregular. The lividity, under these circumstances, depends on congestion of the venous radicles, arising from inability of the heart to receive the blood returned to it by the systemic veins. But this symptom generally involves an affection of the pulmonary system, such as pleuritis or pneumonitis, existing in combination with the pericar-

ditis. The impaired ability of the lungs to aerate the blood is then associated with venous congestion in the production of lividity. It is unnecessary to add that lividity is a symptom incident to a variety of cardiac and pulmonary affections, and does not therefore possess any intrinsic significance as diagnostic of pericarditis. It is not, in fact, always present in fatal cases of the latter disease, even when characterized by great liquid effusion. These remarks are equally applicable to the symptom last noticed, viz., œdema.

Symptoms referable to the respiratory system.

In cases of pericarditis disconnected from any affection of the lungs, the respirations are sometimes accelerated in consequence of the inspiratory acts being shortened by præcordial pain. I have observed marked dilatation of the *alæ nasi* under these circumstances. Cough, dry, hacking, or spasmodic, is common, and does not denote, necessarily, coexisting pulmonary disease. Dyspnœa may be an urgent symptom, dependent generally on congestion of the lungs incident to compression of the heart by liquid effusion. These symptoms, however, are extremely variable and inconstant, as are all the symptomatic events belonging to the clinical history of pericarditis. The respiration may be unaffected, or accelerated only in proportion to the febrile movement. Cough is not uniformly present. Dyspnœa may be wanting even when the pericardial sac is largely distended. Moreover, each of these symptoms occurs in a variety of pathological relations, and is not, therefore, distinctive of pericarditis. They are often dependent, to a greater or less extent, on coexisting pleurisy or pneumonia. When these affections are excluded, dyspnœa denotes, as a rule, obstruction to the circulation caused by weakness of the heart or mechanical compression, or both combined. As thus produced, it may exist in a degree to constitute orthopnœa. It belongs, in general, to the second stage of the disease, and is associated with frequency and feebleness of the pulse, and perhaps with lividity of the prolabia and face. Under these circumstances, it denotes imminent danger. Cases, however, have been observed in which dyspnœa, and even orthopnœa, existed at the commencement of the disease, prior to effusion, and when it was not attributable to any coexisting pulmonary affection. In some instances, as suggested by Dr. Sibson, the distended pericardial sac may add to the dyspnœa by pressing on the trachea at its bifurcation. The augmented space which the pericardial sac

occupies when largely distended also contributes to the production of dyspnœa.

The voice has been observed to be remarkably weak, the patient being unable to speak in feeble tones without considerable effort. Dr. Walshe, who has noticed this symptom, states that it seems to be mainly connected with copiousness of effusion.

Symptoms referable to the digestive system.

Certain cases of pericarditis are characterized by prominent symptoms referable to the digestive system, but, occurring only occasionally, they are incidentally connected with the disease, and can hardly be considered as forming a part of its clinical history. Thus, vomiting is sometimes present and persisting in a marked degree. Dr. Copland has remarked that, under these circumstances, there is some liability to mistake the disease for gastritis, the rapid, weak, irregular pulse, etc., being attributed to a tendency of the latter affection to an unfavorable termination. Dysphagia is another symptom belonging in this category. Its occasional occurrence in cases of pericarditis was noticed by an Italian author, Testa, who published a work on diseases of the heart in 1811.[1] Dr. Stokes and Dr. Walshe have observed it in several cases. It has not been ascertained to depend on any appreciable alteration in the pharynx or the adjoining parts, and is therefore to be regarded as either a spasmodic affection, or a mechanical effect of pressure of the distended pericardial sac upon the œsophagus.

These are the only symptoms to be mentioned under this head. Loss of appetite, thirst, constipation, etc., are incident to pericarditis as well as to acute inflammation affecting any important organ, and accompanied by febrile movement.

Symptoms referable to the countenance, position, etc.

An expression of anxiety or apprehension is frequently a marked symptom, as it is in cases of merely functional disorder of the heart. In severe cases, near the fatal termination, the *risus sardonicus* has been observed. Lividity and œdema are occasional symptoms which have been already mentioned.

The position assumed by the patient is generally on the back;

[1] *Vide* Stokes on Diseases of the Heart and Aorta, Am. ed., p. 69.

or he takes a diagonal position between that on the back and on the side. He rarely lies on the left side, the liver in this position pressing upon the heart, and thus giving rise to discomfort or adding to the distress. In some cases a position on the right side is not uncomfortable. If pleurisy or pneumonia coexist, the decubitus will, of course, be in a measure determined by these affections. Generally, but not invariably, the patient desires to have the head and shoulders raised.

All observers have noticed this point relating to position, viz., whatever may be that selected by the patient, he is reluctant to change it; *i. e.*, he desires, as much as possible, to maintain the same position. This is accounted for by the fact that movements of the body increase distress, and, by exciting the heart, give rise to a sense of syncope, especially when the pericardial sac is distended with liquid. Fatal syncope may be induced by a change of position. I have known death to occur suddenly, when a fatal termination was not expected, apparently being caused by the patient rising from bed and going to stool.

When the amount of liquid effusion is large, a recumbent position on the back may still be preferred; but instances have been observed in which patients have experienced relief from lying on the face. If dyspnœa be urgent, a sitting posture may be alone tolerable, the dyspnœa then constituting orthopnœa. Under these circumstances, restless movements of the arms are common, the body remaining comparatively immovable.

Symptoms referable to the nervous system.

Mental aberration, moderate or slight, and transient, is not uncommon in cases of pericarditis. I have known it to occur at the commencement of the disease, and soon disappear, the patient afterward preserving the faculties of the mind to the close of life. It is oftener observed at a later period in fatal or severe cases. It is not, however, an element of the disease. In many, perhaps in the majority of cases, it is wanting. But in certain cases of pericarditis, cerebral symptoms are developed which are highly important in themselves, and also because they serve to mask the local symptoms of cardiac disease. The cerebral symptoms now referred to, resemble those which characterize different affections of the nervous system. Inflammation of the meninges of the brain, mania, dementia, coma, epilepsy, tetanus, and chorea have been simulated in

cases of pericarditis, the latter disease being generally overlooked before death, and examination post-mortem revealing no appreciable lesions of the brain or spinal cord adequate to explain the phenomena observed during life. The phenomena in these remarkable, and, as they have been justly called, fearful, cases must needs be diversified in order to give rise to a resemblance to each of the several affections just named; yet there are certain features which are somewhat distinctive. This subject has been considered more fully by Dr. Burrows than by any other author within my knowledge.[1] Dr. Burrows gives a synopsis of all the cases that he was able to gather from various sources. It is remarkable that the subject is merely alluded to in works devoted specially to diseases of the heart. Without detailing the cases which have been reported, I shall refer to them sufficiently to present a sketch of the varied symptoms which they embrace; and I shall add a brief account of three striking cases which have fallen under my own observation. So much, the importance of the subject seems to me to demand.

The first recorded case, according to Dr. Burrows, was reported by Dr. Stanley in 1817. The next case was communicated by Dr. Abercrombie in 1821. Dr. Latham reported a case in 1828; and he states that "when he first related the particulars of his case to several medical friends, they looked incredulous, or rather contemptuous of the man who would mistake an inflammation of the pericardium for an inflammation of the brain." In each of these cases the patient was supposed to labor under a cerebral affection, to which the treatment was directed, and the existence of cardiac disease was not suspected prior to the autopsical examination. The brain presented no evidence of disease beyond a certain amount of congestion. Other cases were subsequently reported by Andral, Bouillaud, Copland, McIntosh, McLeod, Hawkins, Bright, Watson, and others. Dr. Burrows states that not less than six cases have come under his own observation. Of 16 recorded cases cited by the author last named, 11 proved fatal, and only 5 recovered. Of the 11 fatal cases, in 2 only was an affection of the heart detected during life; in 1 cardiac disease was suspected, but in the remaining 8 cases there was no suspicion of an acute affection of the heart until it was revealed by an examination after death. Of the 5 suc-

[1] On Disorders of the Cerebral Circulation, and on the Connection between Affections of the Brain and Diseases of the Heart. By George Burrows, M. D., etc. Am. edition, 1848.

cessful cases, in 4 the diagnosis of cardiac disease was satisfactorily established. A rational inference from these facts is, that when the existence of cardiac disease is ascertained, the cases are usually amenable to treatment; but, on the other hand, when the cardiac affection is overlooked, a fatal result usually occurs.

In the sixteen cases detailed in Dr. Burrows' work, were manifested, delirium, convulsions, agitation of the limbs resembling chorea, a state of dementia, a species of coma, seizures resembling apoplexy, and tetanic spasms. The delirium was characterized by taciturnity, and maniacal excitement under the influence of delusions involving the idea of having committed some crime. Convulsions occurred in paroxysms, and the choreic form was accompanied by rolling of the eyes and head, as well as violent agitation of the limbs. The coma was characterized by the eyelids remaining open, and the eyes fixed. In the apoplectiform seizures, the eyeballs were turned upward and the limbs paralyzed. In several instances, violent tonic spasms occurred, resembling tetanus. In the fatal cases, death was generally preceded by ordinary coma.

The first of the cases which have fallen under my observation, occurred in 1849, and was reported by me, February, 1850.[1] The patient was admitted into the hospital in a state of active delirium, and nothing was ascertained respecting the previous history. On the following day he was tranquil, and, when spoken to, made no reply, shaking his head. The eyes had a wild, staring expression. He could not be made to protrude the tongue. Pulse small, feeble, and not accelerated. Active delirium occurred at intervals, during which he shouted and cried, as if from apprehension of danger. At other times he lay with his eyes open and fixed in a particular direction, taking no notice of persons and things around him. On several occasions he answered questions, and he then gave evidence of the delusion that he had committed some crime. Once, when asked how he was, he replied, "Guilty." At another time he had an impression that he was confined in jail, and subsequently he asked why he had not been hung, etc. On the fourteenth day after his admission, he complained, for the first time, of pain in the chest, and, on physical examination, the signs of pleuro-pneumonia were discovered. There were no cough and expectoration. Death occurred on the seventeenth day. Delirium with hilarity occurred on the last day, and he became comatose for several hours before

[1] Buffalo Medical Journal, vol. v. p. 505.

death. On examination post-mortem, the left lung was in the second stage of inflammation, and the pleural sac contained about twelve ounces of turbid serum. The surface of the heart was covered with recently exuded lymph. The endocardial membrane was healthy and the valves sound. The brain presented no other evidence of disease than a considerable amount of congestion, and slight opacity of the arachnoid over the superior surface of the cerebrum.

The existence of pericarditis was not suspected, in this case, prior to the autopsy. Up to the fourteenth day, the affection was supposed to be exclusively cerebral, no symptoms pointing to the chest as the seat of the disease. Physical exploration was neglected until the date just stated, when pneumonia was ascertained.

The second case came under observation in the hospital at Buffalo in 1851, and was reported by me in January, 1854.[1] At my morning visit, I found that on the previous evening a patient had been admitted greatly prostrated and delirious. Nothing was obtained relative to the previous history. The patient had not spoken since his admission. He lay with his eyes open, fixed, most of the time, in one direction, taking no notice, and making no reply to questions. A disagreeable peculiarity in this case was, that the patient frequently ejected saliva with force, and without any regard to its destination. His bed and the floor were bespattered with spittle. Persons in proximity to him were liable to receive it on their persons, not from design, but because it was scattered at random, the patient not changing his position and lying on his back. Under these circumstances, an examination of the case was deferred, and at my next visit I found that the patient had died. At the time I was observing the patient, the idea of pericarditis did not occur to me, but in thinking of the case afterwards, a resemblance in the character of the delirium to that in the preceding case, led me to suspect this disease; so that, before the autopsy was made, I ventured to predict that it would be discovered. My prediction proved true. The pericardium was universally adherent by recent tender adhesions. Pulmonary disease did not coexist in this case.

The foregoing cases ended fatally. The third case came under observation in the Louisville Marine Hospital, October, 1853, and terminated in recovery. This case was reported in connection with the preceding case. When admitted, the mind of the patient

[1] Buffalo Medical Journal, vol. ix. p. 449.

was too dull to give any connected account of past or present symptoms. On the day following he was delirious, frequently getting out of bed, and seemed bewildered. The next day he was unconscious. He lay upon the back, taking no notice of persons and things around him. He had lost one eye; the other remained open, and the pupil was dilated. He was taciturn, and could not be roused to reply to questions; he urinated in bed; the saliva escaped from the mouth, and he did not swallow when drink was introduced. The physical signs, exclusive of friction-sounds, were sufficient to establish the existence of pericarditis with moderate effusion.

Convalescence was established a fortnight after his admission. For three days he took neither drink nor nourishment, making no effort to swallow, and sometimes resisting their introduction by forcibly closing the teeth. At times during the three first days he was exceedingly restless, throwing himself from the bed, so that it became necessary to transfer him to the floor. The pulse was 80, and the respirations 28. On the fourth day, in the morning, there was marked improvement. The patient took food and drink, and appeared to notice objects around him. This peculiarity was observed, viz., he directed his vision to some point, now a portion of the pillow and now his hand, protruded his tongue towards it, and then slowly grasped it with his lips and teeth. This he repeated frequently. In the course of the day he again became restless, throwing himself about, getting up, calling names of different persons. The day following, his expression was idiotic. His eye was open, and he looked about with a vacant stare. He resisted physical exploration. Twice he said while the record of symptoms was being made, "I beg pardon." These words were uttered spontaneously, with slowness and hesitancy. He did not reply to questions, and was taciturn the greater part of the time. On the sixth day he had three attacks of convulsions an hour in duration. These recurred on the day following. In the intervals he frequently got out of bed and endeavored to break the walls of the room, as if to escape from persons threatening violence. It was necessary to apply a restraining jacket. On the eighth day he lay night and day rolling about the floor and shouting incoherent words. On the tenth day he continued wakeful, shouting, with occasional manifestations of hilarity. In the course of this day he slept quietly for several hours, took food and drink readily, protruded the tongue, and replied to questions. From this time he

was rational, and, on being questioned, said that he had pain in the left breast above the nipple, lancinating, and increased by deep inspiration. He had no recollection of the events of the previous fortnight. He stated that he was ill for two days before coming to the hospital, and that he suffered chiefly from pain in the left breast. He convalesced from this date.

The physical signs on which the diagnosis was based in this case, were flatness on percussion over an increased, pyramidal space in the præcordia; elevation of the point of apex impulse and flatness below this point; irregularity and feebleness of the heart's contractions; diminution of the area of præcordial dulness at the time of convalescence, and retraction of the intercostal spaces with the heart's action. Friction-sounds were not discovered, but the early application of a blister over the præcordia interfered somewhat with auscultatory exploration. In connection with the physical signs denoting pericarditis, pain referred to the præcordia was a prominent symptom before the patient entered the hospital, and was felt after his consciousness returned, together with tenderness in the same region, evidently not dependent on the blister. Acute pulmonary disease was excluded by the absence of physical signs, as well as symptoms, pertaining to the lungs.

It is worthy of remark that in the three cases of pericarditis associated with cerebral disorder, of which an account has just been given, the disease in each was not developed during the course of rheumatism. Of the sixteen cases analyzed by Dr. Burrows, in seven no rheumatic affection could be discovered. In the third case the pericarditis seemed to be purely idiopathic, and in the two other cases no antecedent affection was ascertained.

Although the manifestations of cerebral disorder incident to pericarditis are so varied, there are certain points of resemblance in the different cases, and, on the other hand, dissimilarity in certain respects from disorder occurring in other pathological relations. The variety of manifestations occurring in the same case, is somewhat distinctive. Different forms of delirium, coma, convulsions, etc., are developed successively during the progress of the disease. The characters pertaining to the delirium are peculiar; the patient lying in a species of coma vigil, the eyes open and fixed in one direction, not replying to questions, and incapable of being roused; this state followed by maniacal excitement, the patient shouting and apparently laboring under the fear of harm, with occasional ebullitions of hilarity. A fixed delusion of having committed some

crime, appears to be a distinguishing feature. Meningeal inflammation does not give rise to this sort of delirium. Moreover, the acute pain in the head, throbbing of the carotids, injection of the eyes and face, which belong to the symptomatology of acute meningitis, are wanting. The delirium offers but a faint resemblance to that of delirium tremens, and to that which is distinguished from the latter as delirium ebriosum. It has no resemblance to the quiet, muttering delirium of continued fever, nor the active delirium which sometimes occurs in the course of febrile diseases. These points, if not diagnostic, should, at all events, excite strong suspicion of the existence of pericarditis in the absence of any symptoms pointing directly to the heart as the seat of disease. The diagnosis in such cases must rest on the presence or absence of physical signs denoting inflammation of the pericardium.

The symptoms referable to the nervous system which have been considered, are, happily, infrequent. But instances of their association with pericarditis are sufficiently numerous to show some pathological connection between the latter disease and the nervous disorder. The complication is not due merely to coincidence. The instances, in fact, it is probable, are more numerous than would be inferred from the number which have been reported. As remarked by Dr. Burrows, how many have occurred in the practice of physicians who have been less candid than Drs. Abercrombie, Latham, and others, in recording their mistakes, and how great a number must have happened in the practice of those who were unable, or took no pains to distinguish these deceptive cases, it is impossible to say. It is, however, but fair to add, that some observers of large experience, who have given special attention to diseases of the heart, have not met with examples. Dr. Hope, at the time of the publication of the second edition of his work, had not observed an instance, and Dr. Stokes, in his recent work, alludes to the subject in terms which imply that an instance has not fallen under his observation. The author last named considers the connection between the nervous disorder and pericarditis as doubtful.

Of the nature of the pathological connection assumed to exist between the nervous symptoms and pericarditis, but little is to be said. The former are not dependent on any appreciable morbid conditions of the brain or spinal cord. They are, therefore, with our present knowledge, to be regarded as functional. They have been attributed to disordered circulation, an altered state of the blood, and nervous irritation transmitted through the phrenic and

pneumogastric nerves. Perhaps the most rational view is, that they proceed from the same general conditions which give rise to the associated pericarditis; in other words, that the connection is simply one of a common causation.

In addition to the symptoms which have been considered, there are none of importance, referable to the nervous system, remaining to be noticed. Pain seated in or near the præcordia, is included among the symptoms relating directly to the heart. Sleep is often more or less impaired by pain or dyspnœa. As regards the state of the mind, anxiety, apprehension, and mental depression belong to the history of the disease, and are frequently prominent symptoms.

The genito-urinary system offers no symptoms which have special relations with pericarditis. Albuminuria coexists in a pretty large proportion of cases, and is an important symptom of a morbid condition of the kidneys, antecedent to the pericarditis, and upon which the development of the latter depends. Under these circumstances, it is not properly a symptom of the cardiac disease.

Physical Signs of Acute Pericarditis.

In treating of the symptoms of acute pericarditis, the absence of distinctive characters derived from this portion of the clinical history of the disease, has been apparent. The deficiency, as regards diagnostic points, in the symptomatic events which have been considered, enhances the importance of the physical signs. In fact, it is mainly by means of the latter that the disease may be now generally recognized with a degree of positiveness which clinical observers, but a few years ago, regarded as unattainable. Of the several methods of exploration, all, save succussion, furnish signs of more or less value. I shall consider the signs obtained by percussion, auscultation, palpation, inspection, and mensuration, respectively, under separate heads, in the order in which these different methods are now enumerated.

Signs furnished by percussion.

The signs furnished by percussion in pericarditis, are due to the accumulation of liquid effusion. The value of this method of exploration consists in the information which it affords as regards

the presence or absence of liquid, the amount of distension of the pericardial sac, the variations in the quantity of liquid during the progress of the disease, and its final disappearance.

Accumulation of liquid within the pericardial sac increases the area and the degree of præcordial dulness, and renders the sense of resistance in practising percussion greater than in health. Effects similar to these are produced by enlargement of the heart. The practical inquiry, therefore, arises, what circumstances distinguish the increased extent and amount of dulness incident to the presence of liquid, from the same effects as produced by cardiac enlargement?

The pericardium is a pyriform sac, the lower extremity forming the base. It extends upward above the base of the heart, rising as high as the cartilage of the second, and sometimes of the first rib. Unaffected by disease, it is capable of holding, as determined by Dr. Sibson's experiments already referred to, from 15 to 20 ounces of liquid. The effusion in cases of acute pericarditis rarely much exceeds this amount, although the quantity is vastly greater in some cases of the chronic form of the disease. The area of dulness, when the sac is distended with liquid effusion, corresponds, not only to the increased size, but to the form and situation of the sac. In acute pericarditis the sac does not undergo any marked alteration in shape or dimensions. It forms, when enlarged to its full capacity, a pyriform tumor extending from the junction of the cartilage of the second or first rib with the sternum, on the left side, downward to the sixth rib or intercostal space. Laterally, in proportion to the distension, it pushes aside the lungs, increasing the space caused by the divergence of the right and left lung, called the superficial cardiac region. If the liquid be not sufficient to distend the sac, it gravitates to the lower part, and the dilatation advances from the base upward in proportion as the accumulation goes on. After a certain amount of accumulation has taken place, the heart is raised upward, and the base of the sac somewhat depressed, so that the apex of the organ is situated at a distance from the bottom of the sac, the space below the apex being, of course, occupied by liquid.

The foregoing points, relating to the physical conditions which belong to the period of effusion in pericarditis, are involved in the circumstances distinctive of the signs furnished by percussion in this disease. The most distinctive circumstance is that already

mentioned, viz., an area of præcordial dulness corresponding to the pyriform shape of the pericardial sac, commencing at the sixth costal cartilage or intercostal space, and extending upward to the second or first rib. If the sac be distended to nearly or quite its normal capacity, and no other morbid conditions are present to obscure the physical signs, its situation and shape may be delineated upon the chest with less difficulty than the space occupied by the heart in cases of enlargement, in consequence of the dulness and sense of resistance being more marked. In cardiac enlargement, the area of dulness does not present the pyriform shape which characterizes that due to pericardial effusion. It corresponds to the form and situation of the heart. The area is extended chiefly in a lateral direction below the third rib, and especially to the left of the sternum, whilst the extension is vertical rather than horizontal when the pericardial sac is distended with liquid. The distinction is marked if the accumulation of liquid be sufficient to distend the sac. It is less so, if the sac be but partially filled. The area of dulness is then widened from the base of the præcordial region, upward, to a greater or less extent, in proportion to the quantity of liquid. This lateral enlargement, however, is arrested, after reaching a certain distance, and, as the accumulation goes on, the dulness extends upward above the normal boundary of the base of the heart. When the pericardial sac is filled, the situation and shape of the area of dulness, as determined by percussion, are almost sufficient for the diagnosis; but if the sac be but partially filled, the signs obtained by percussion must be taken in connection with those furnished by other methods of exploration. A moderate amount of liquid will widen the area of dulness. Dr. Walshe states that four ounces are sufficient. To be appreciable, however, by percussion, when the sac is but partially filled, the præcordia must have been examined before the effusion occurred, and the fact of enlargement thus determined by comparison. The degree of dulness and sense of resistance are to be taken into account. The dulness when the pericardial sac is distended, may amount nearly, or quite to flatness, and the elasticity of the ribs is diminished in a notable manner. These effects are much more strongly marked in cases of chronic pericarditis with large effusion.

A circumstance highly distinctive of liquid accumulation in pericarditis, is the variation in the extent of dulness at different periods during the course of the disease. Effusion often taking place rapidly, if the præcordia have been examined prior to its

occurrence, a remarkable enlargement of the area of dulness is sometimes observed after the lapse of a few hours. This enlargement may be found to have been progressive at successive examinations until it reaches a certain extent, where it may remain stationary for a greater or less period, and then decrease more or less rapidly. Fluctuations from day to day are not infrequently observed, the extent of dulness now increasing and now diminishing, until, at length, if the termination of the disease be favorable, it is reduced to the normal limits. On the other hand, the dulness from cardiac enlargement is not developed thus, as it were, under the eyes of the observer. Its extension is so gradual as, in general, to be imperceptible on comparative examinations made after intervals of weeks and even months. It never fluctuates, nor diminishes in extent. In cases of pericarditis, the daily employment of percussion is highly useful in determining not only the existence, or otherwise, of effusion, but its increase, diminution, and final disappearance. Information concerning these points may influence considerably the treatment of the disease, as well as the prognosis.

Another distinctive circumstance is derived from the relation of the area of dulness to the point of apex-beat of the heart. In cases of cardiac enlargement, the beat of the heart is felt in the lower limit of this area. This limit is often defined by tympanitic resonance due to gas in the stomach. In cases of considerable pericardial effusion in which the apex-beat is felt, it is raised above the lower border of the area of dulness from the presence of liquid. Percussion below the apex-beat yields a dull or flat sound for a certain distance, before a gastric tympanitic resonance is reached. This test is available in some cases, but not invariably. It requires that the apex-beat shall be appreciable either by the eye or touch, and the presence of gas in the stomach. The percussion should be light or superficial.

The signs furnished by percussion in pericarditis, as already stated, relate to the effusion of liquid incident to the disease. Prior to effusion, and subsequently, this method of examination does not afford important information except in a negative point of view. The presence of lymph, it is true, increases somewhat the size of the heart, but not to an extent to give rise to an appreciable enlargement of the area of dulness. The availability of these signs when the pericardial effusion is abundant, may be impaired or destroyed by the coexistence of other morbid conditions affecting the adjacent parts. If the pericarditis be accompanied by pleurisy

with effusion, it is difficult, or impossible, to define the dulness due to the dilated pericardium. Difficulty and error, were the signs furnished by percussion to be exclusively relied upon, would be likely to arise, as indicated by Dr. Walshe, from an aneurismal sac at the arch of the aorta, or a small mediastinal tumor, or even a superabundance of fat just above the third left cartilage, all of which, in connection with enlargement of the heart, may simulate the pyramidal form of the area of dulness which is characteristic of distension of the pericardial sac.

Signs furnished by auscultation.

The auscultatory phenomena in cases of pericarditis, are of great importance in their relations to diagnosis. It is more especially with respect to these, that the knowledge acquired within late years has been so useful in enabling the physician to determine, with positiveness, the existence of the disease. The signs furnished by auscultation relate, *first*, to the development of new or adventitious sounds, generally known as attrition or friction-sounds; *second*, to abnormal modifications of the heart-sounds; and, *third*, to respiratory and vocal sounds in proximity to the præcordial region.

The sounds of attrition or friction are also called exocardial, pericardial, and peripheral sounds. The term friction-sound is the most simple, and sufficiently expressive. The occurrence of a friction-sound in pericarditis was first noticed by a French observer, M. Collin. The sound observed by him, he compared to the creaking of new leather (*bruit de cuir neuf*), and he attributed it to an unnatural dryness of the inflamed pericardium. A sound resembling the crackling of parchment was subsequently observed by Broussais. But the inauguration of friction-sounds as constituting a frequent physical sign of pericarditis, is to be dated from a publication by Dr. Stokes in 1834. The attention of Dr. Watson, of London, was directed to these sounds simultaneously with Dr. Stokes, and they were shortly afterward observed by Bouillaud, without his being aware of the prior observations of his British co-laborers.

The motions of the heart in its contractions and dilatations, but more particularly its movements of rotation, involve friction of the opposed pericardial surfaces. But in the normal condition of the membrane, a friction-sound, as a rule, is not audible, exclusive of the element of impulsion in the first sound, which has been de-

scribed in Chapter I. of this work. This element in some persons has somewhat of a friction character; and I have occasionally discovered, in auscultating the heart, a slight rubbing or grazing sound accompanying the systole, when there were no grounds for suspecting any cardiac disease. These exceptions to the general rule do not impair the practical value of the physical sign as an indication of disease. The physical conditions necessary for the production of a morbid friction-sound, are due to the products of inflammation deposited upon the pericardial surfaces, by which they are roughened and caused to adhere, instead of gliding smoothly and noiselessly upon each other. These products differ in different cases, and in different periods of the disease in the same case, as regards abundance, density, disposition, etc. The character of the friction-sound is doubtless affected by these differences, but it is difficult or impossible to determine for each variety of sound a special significance, by which may be recognized, with precision, corresponding variations in the physical conditions. Like endocardial murmurs, the sounds are sometimes rough, and sometimes comparatively soft. The latter convey the idea of a gentle grazing or rubbing; the former are distinguished as grating, scraping, or rasping sounds, and denote a more abundant and dense deposit of lymph. The creaking sound described by Collin is attributed to the stretching of lymph which has led to partial adhesions. Dr. Walshe thinks that it may be produced when the surfaces are so closely agglutinated that attrition or separation is physically impossible, being caused by the bending and crumpling of tough, false membranes. Occasionally, according to this observer, the sounds have a clicking character. A continuous rumbling sound is sometimes heard, which is supposed to be due to the presence of a small quantity of liquid with which soft lymph is commingled. A churning or splashing sound occurring after a penetrating wound of the chest, causing a small aperture into the pericardium, but without giving rise to pericarditis, was described to me by Dr. Knapp, of Louisville, due probably to the escape of a small quantity of blood into the pericardial sac. To attempt to describe all the varieties of friction sounds, and apply to them different names, would render the subject needlessly complicated. It is important to know the diversities of character which they assume, only so far as this knowledge is instrumental in aiding in their recognition at the bedside.

A friction sound may accompany the systolic and diastolic

movements of the heart, separately or combined. In the majority of instances it accompanies both movements, or, in other words, is double. Hence, it was described by Dr. Watson under the name of the "to-and-fro rubbing sound." When single, it is generally systolic. A diastolic friction-sound occurring alone must be exceedingly rare. A double sound may be heard in certain situations, and only a single sound in other situations. The intensity varies in different cases, and even at different periods in the same case, within wide limits. In some instances so faint as to be discovered only with the closest attention, it is, in other instances, sufficiently loud to be heard with the ear removed at a short distance from the stethoscope. Between these extremes there is every gradation of intensity. The systolic friction-sound may be intense, and the diastolic comparatively feeble. The converse of this must be rare. The intensity will depend, in a great measure, upon the physical conditions which give rise to the sound; but also upon the force of the heart's movements. Variation, in the latter respect, at different times in the same case, will affect the intensity of the sound. Bleeding and debilitating remedies, by weakening the heart, have been observed to lessen the intensity in a marked degree. On the other hand, the intensity is notably great when pericarditis supervenes on hypertrophy of the heart.

Under what circumstances are friction-sounds developed within the pericardium, in cases of pericarditis? In order for their production, the visceral and parietal surfaces must, of course, come into contact during the movements of the heart. The accumulation of liquid in the pericardial sac, separates these surfaces to a greater or less extent, but does not necessarily prevent them from coming into contact. The movements of the heart may bring these surfaces together at certain points, and, in a recumbent position, the organ naturally gravitates to the bottom of the liquid, and rests upon the depending portion of the sac. Hence, friction sounds are by no means uniformly arrested by an abundant liquid effusion. They are observed in some cases in which the quantity of liquid is extremely large. In many, perhaps in the majority of cases, however, they do disappear during the period of effusion, which is probably owing to weakness of the heart's action, and not to an entire separation of the pericardial surfaces. The sounds are most likely to be produced anterior and subsequent to the period of effusion. They may be developed very soon after the commencement of the disease. Dr. Walshe states that friction was detected in a case in

which fatal perforation of the œsophagus and pericardium was produced in the attempt to swallow a sword, thirty minutes after the accident. Here, the sound was probably due to the presence of blood in the pericardial sac. I have observed it well marked six hours after the sudden occurrence of pain, etc., denoted an attack of pericarditis. It is rare that patients are seen at an earlier period in the disease. In that case, the disease was developed in hospital, in connection with Bright's disease. It has been supposed to occur prior to the exudation of lymph, and to be dependent on dryness of the membrane. This must be considered as conjectural. Dr. Walshe states that he has known mere vascularity of a very small surface, without a particle of lymph, to produce a faint rubbing noise. This, however, is sometimes observed when the membrane may be presumed to be entirely healthy. As a rule, to which there are very few exceptions, the presence of a friction-sound implies deposit of lymph in more or less abundance. Moreover, as a rule, a friction-sound is developed whenever exudation of lymph takes place, and continues up to the period of effusion, if not into this period. The value of the sign in diagnosis depends, in a great measure, on this important fact. Clinical observation shows that the absence of a friction-sound in pericarditis, if auscultation be employed with care from an early period of the disease, is a rare exception to the general rule. When not observed, assuming proper care and ability in the observer, it is probable that, in most instances, it existed prior to the case coming under observation. It may soon disappear, after becoming developed, in consequence of weakness of the heart and liquid effusion. Dr. Walshe states that he has known it to appear and to disappear finally, within the space of six hours. If it disappear during the period of effusion, it often returns after the liquid is absorbed, and the pericardial surfaces again come freely into apposition. This returning friction-sound, in conjunction with the physical signs obtained by percussion, becomes evidence of the removal of the liquid, and is, therefore, of favorable omen. It is, however, less constant at this period than during the period preceding effusion, in this respect differing from the friction-sounds incident to pleuritis, the latter being developed much oftener after, than before the stage of effusion. The character of the sound developed in the third period of the disease may differ from that in the first period. If, prior to effusion, it was rubbing, grazing, or churning, it may become, after absorption, rasping, grating, or creaking. Adhesion of the pericardial surfaces

generally arrests the sound, but this rule is not invariable. Stretching of newly-formed tissue and bending of the exudation matter, may give rise to a creaking sound. The disappearance of the sound may be abrupt, or gradual, generally the latter. Like pleural friction-sound, it may continue for a considerable time after convalescence and apparent recovery. Dr. Walshe refers to an instance, under his observation, of its persistence for three months, continuing long after the patient's discharge from the hospital, and when he seemed to be perfectly restored to health.

Friction-sounds are to be discriminated at the bedside from endocardial murmurs. According to the testimony of numerous observers, this is sometimes extremely difficult. The two are often confounded by those who assume to employ physical exploration, with an imperfect knowledge of the subject. But with proper care, and an acquaintance with the differential points involved in the discrimination, the instances are rare in which a practical auscultator is much embarrassed. The distinctive circumstances are important to be considered. In many instances the intrinsic character of the sound is sufficient, in itself, to mark the distinction. The sound conveys to the mind the idea of the rubbing together of rough surfaces. This, however, is by no means to be relied upon to the exclusion of other distinctive circumstances. A rough valvular murmur sometimes simulates closely, so far as the character of the sound is concerned, a friction-sound. Other points are to be taken into account. Friction-sounds are generally double, *i. e.*, systolic and diastolic. This is a point of minor importance, since double endocardial murmurs are not very infrequent. Points of much greater importance relate to the localization and diffusion of friction-sounds considered relatively to endocardial murmurs. Friction-sounds are usually limited within the space occupied by the heart. They very rarely extend beyond the borders of the organ. In general, they abruptly cease when the stethoscope is removed but a short distance without the boundaries of the heart, although they may be quite intense everywhere within the limits of the cardiac region. This is certainly the rule, but exceptions undoubtedly do occur. With endocardial murmurs the rule is otherwise. These are generally heard with greater intensity at points removed from the heart, than over the heart itself. Aortic murmurs are loudest above the base of the organ, in the second intercostal space. Mitral murmurs are usually most intense to the left of the apex. Both mitral and aortic murmurs are often dif-

fused to a considerable distance from the heart, the latter alone as far as the carotids, and the former not infrequently over the left lateral and posterior surfaces of the chest. The maximum of intensity of pericardial friction-sounds is generally over the body of the heart, within the superficial cardiac region. They are frequently limited to this region, or even to a portion of it, which may be either towards the base or apex, oftener the former when they are thus circumscribed. It is only in some rare instances that endocardial murmurs are heard exclusively within the superficial cardiac region. This, however, is sometimes the case with murmurs which I have called intra-ventricular, as distinguished from those produced at the orifices.[1] These points are highly distinctive. Friction-sounds do not observe rhythmical relations to the heart-sounds as do endocardial murmurs. An endocardial murmur has a certain connection with one of the sounds of the heart, and this connection is uniformly maintained. A friction-sound, on the other hand, frequently seems to occur discordantly, as regards the heart-sounds, varying in rhythm irrespective of the latter. Variableness is a distinctive trait of friction-sounds, as compared with endocardial murmurs. The latter undergo but little change with successive beats of the heart; while the former vary, even during the time occupied in an examination, as regards intensity, the occurrence of one or two sounds with a single beat, the character of the sounds as regards roughness and softness, the situation in which they are loudest, etc. Their variableness, as regards duration, is distinctive. Endocardial murmurs are generally persisting. The latter are rarely affected materially by change in the position of the patient. Friction-sounds are not only more intense in certain positions than in others, but they are sometimes heard only when the body is in a particular posture. Thus, they may be apparent when the patient lies on the back, and disappear when he is sitting, or *vice versâ*. Their intensity may be increased or diminished by inclining the body backwards or forwards. The explanation of this is, that in certain positions the pericardial surfaces are brought into contact at points where the physical conditions are most favorable for the production of sound. These variations in different positions are observed more especially when more or less liquid effusion is contained within the pericardial sac. Friction-sounds may be discovered by auscultating in different

[1] *Vide* Chapter IV. page 202.

positions, when, were the examination limited to one position, they would fail to be apparent. Another distinctive circumstance is their apparent proximity to the ear of the auscultator. They seem to be superficially seated, resembling often the sound produced by friction of the clothing upon the stethoscope. This resemblance is sometimes so striking that the observer looks to see whether the sound be not actually thus produced. An exception to this rule is, when a friction-sound is produced on the posterior surface of the heart. On the other hand, endocardial murmurs, as a rule, appear to emanate from points more removed from the ear, or situated deeper within the chest. This is a highly distinctive point.

Finally, as indicated first by Dr. Sibson, firm pressure with the stethoscope is found generally, but more especially in young subjects, to intensify friction-sounds. It does this partly by increasing the conduction, and in part by displacing the stratum of liquid, bringing the pericardial surfaces into closer apposition, and rendering the walls of the chest more resisting. In this way, a friction sound which has disappeared in consequence of effusion, may sometimes, according to Dr. Sibson, be reproduced. An effect of pressure is sometimes to change the character of the sound, converting it from a soft to a rough sound. As stated by Dr. Walshe, the pitch may be raised. Endocardial murmurs are not intensified to the same extent by pressure. It is incorrect to say that they are in no degree intensified. Their intensity is augmented in so far as the conduction is increased. It is in this way alone, probably, that pressure intensifies endocardial murmurs. The difference in the result in the two cases, constitutes this an important point of distinction. Attention to the several circumstances just mentioned renders, in most instances, the discrimination between friction-sounds and endocardial murmurs sufficiently easy. It is, however, to be borne in mind that endocardial murmurs and pericardial friction-sounds are frequently present in combination, pericarditis coexisting with endocarditis, or the heart being affected with valvular lesions antecedent to the development of the pericarditis. It has been supposed that the deposit of lymph at the base of the heart and about the large vessels within the pericardium, or the accumulation of a large quantity of liquid, may sometimes give rise, by pressure on the vessels, to endocardial murmur. This is doubtful. The frequent combination of the latter with the physical signs of

pericarditis, is to be explained by the coexistence of endocardial inflammation, or valvular lesions.

Pericardial friction-sounds are to be discriminated, at the bedside, from those produced by the rubbing together of the pleural surfaces. Both pleural and pericardial friction-sounds may be combined, since pleuritis and pericarditis are frequently associated. The pleural sounds caused by the respiratory movements, are readily distinguished from the pericardial, by the difference in rhythm. A simple expedient serves to remove any doubt as to their dependence on the action of the heart or lungs, viz., causing the patient to suspend, for a moment, the acts of breathing; if the sounds persist, they are of cardiac origin.

But pleural friction-sound may be produced by the action of the heart. The friction here is *without*, instead of *within* the pericardial sac. Dr. Addison, Dr. Stokes, and others, have reported cases exemplifying the occasional production, in cases of pleuritis, of a *cardiac pleural friction-sound*, which may lead the auscultator into the error of supposing that pericarditis exists when there is no cardiac disease. I can bear testimony to this liability to error. In a case of pleurisy, with considerable effusion, which came under my observation in the New Orleans Charity Hospital, a well-marked, pretty intense, and rough cardiac friction-sound was apparent in the præcordia. It continued when the respiratory movements were suspended. It existed within an area from three to four inches in diameter, and was noted to extend somewhat beyond the left border of the heart. It varied in intensity with different beats of the heart, and with occasional beats was wanting. It was sometimes double and sometimes single, in the latter case being systolic. It was intensified by firm pressure with the stethoscope. It was most marked at the end of a deep inspiration. This sound continued for several days, and was listened to by a large number of physicians and students, who were accustomed to visit the ward, as an excellent specimen of a pericardial friction-sound. Delirium tremens was developed in the case, and the patient died. I expected to find pericarditis associated with the pleurisy, but, on examination, after death, the pericardial sac contained a moderate quantity of transparent serum, and the membrane was perfectly healthy. The heart was in all respects normal; the endocardium presented a natural appearance, and no valvular lesions existed. The left pleural sac contained a large quantity of turbid serum. The lung extended downward, in front to the level of the nipple. It was

connected by tender adhesions to the outer surface of the pericardium, and the costal and pleural surfaces were also united by tender adhesions. The friction-sound was evidently a cardiac pleural sound, produced either by the rubbing together of the outer surface of the fibrous sac inclosing the heart against the adjacent lung (which overlaid the heart), or of the opposed pleural surfaces above the heart. In view of this case, I am prepared to concur in the opinion of Dr. Addison, that "auscultation will not always enable us to distinguish a friction-sound produced *within*, from a friction-sound produced *without* the pericardial sac."[1] Walshe gives the following as the circumstances which argue in favor of friction of cardiac rhythm being of pleural, and not of pericardial origin: "The limitation of the sound to either edge, generally the left, of the cardiac region; fixity in one or more particular spots; cessation complete, or, what is more common, occasional, with certain beats of the heart, when the breath is held; and marked unsteadiness in the intensity and quality of the friction-sound." These circumstances were not fully available for the discrimination in the case which I have cited. It may be added to the account of the case that, in making clinical remarks, attention was called to the occasional occurrence of a cardiac pleural friction-sound, but I confess that I did not regard the case as furnishing an illustration till the chest was examined after death. The sound was not limited to the edge of the cardiac region, but was heard over an area of from three to four inches in diameter. It extended, however, beyond the left border of the heart. It did not cease when the breath was held, but it was most marked at the end of a full inspiration, while pericardial friction-sounds are usually most marked at the end of the act of expiration. "Unsteadiness in the intensity and quality of the friction-sound" certainly characterizes a true pericardial friction-sound in certain cases.

In conclusion, a pericardial friction-sound, as regards diagnostic significance, is, perhaps, the most definite and reliable of all the signs obtained by physical exploration of the chest. Exclusive of cases in which the pericardium is perforated, so that a small quantity of blood escapes into the sac (which is not necessarily followed by inflammation), it is pathognomonic of pericarditis. This can hardly be said of any other physical sign pertaining to the heart or the respiratory organs.

The heart-sounds undergo certain abnormal modifications in

[1] Guy's Hospital Reports, vol. iv. From Bellingham, op. cit.

pericarditis. Early in the disease, prior to the period of liquid effusion, they are either not materially affected, or intensified by the excited action of the heart. An abundant accumulation of liquid occasions marked modifications, more especially of the first or systolic sound. The impulsion of the apex against the thoracic walls being either prevented or greatly weakened, the element of the first sound, which has been designated the element of impulsion, is impaired or lost; the valvular element is left isolated, or it becomes predominant. Hence, the first sound is enfeebled, and shortened, resembling the second sound in quality and duration, but less intense. The second sound is affected only as regards intensity, and, in this respect, much less than the first sound. The second sound, therefore, over the whole præcordia, is the accentuated sound. It is sometimes the only sound discoverable, the first sound being suppressed. The muscular weakness of the heart, arising from compression and the paralyzing influence of the pericardial inflammation, favors these effects. Both sounds appear to be further removed from the ear than in health, in other words, they are more distant. This sense of distance is more obvious when the patient is in certain positions than in others. It is most marked when the patient lies on the back, because the heart is then actually further removed from the anterior walls of the chest. The apparent distance diminishes perceptibly when the sitting posture is assumed, and still more when the patient leans forward. After the removal of the liquid, the normal characters of the heart-sounds return, except so far as they may be modified by weakness of the organ. In these remarks, it is assumed that valvular lesions are not present. These will, of course, be likely to affect the sounds of the heart, irrespective of the pericardits. The abnormal modifications due to the latter, are not without interest; but, as compared with other signs, they are of minor importance.

Auscultation of the respiration and voice may be employed in conjunction with percussion, in order to aid in determining the space occupied by the pericardial sac when distended with liquid. The limits to which the respiratory murmur extends in a direction toward the præcordia, in some cases serves to define the boundary of this space. If these limits coincide with the loss of vesicular resonance on percussion, the two methods mutually confirm each other, as regards the accuracy of their respective results. Vocal resonance is frequently more available for this purpose than the respiratory murmur. It may be found to cease abruptly where

dulness or flatness gives place to vesicular resonance on percussion. These different classes of signs, thus, concur in delineating the line of demarcation between the lungs and pericardium. This application of auscultation presupposes that the lungs are free from disease. Auscultation of the respiration and voice is obviously important in determining the nature and extent of the pulmonary affections which may be associated with pericarditis.

Signs furnished by palpation.

These are important in the diagnosis of pericarditis. Prior to the period of effusion, the beating of the heart is usually found, on palpation, to be abnormally forcible, violent, diffused over a larger area than in health, and sometimes tumultuous. The action of the heart, as thus ascertained, may be in striking contrast with the pulse. Hope enjoins the rule of habitually placing the hand upon the præcordia in all cases of disease, in order that attention may be directed to the heart in instances in which, from the absence of obvious symptoms referable to that organ, cardiac disease is liable to be overlooked. By observing this rule, pericarditis may sometimes be discovered earlier than would otherwise be the case. A practical object in examining the præcordia, by palpation, prior to effusion, is enforced by Walshe, viz., to determine the situation of the apex-beat with reference to subsequent comparisons. When effusion takes place, the apex of the heart is raised and carried to the left. The point of apex-beat which, prior to effusion, was situated in the fifth intercostal space, may be found, after effusion has occurred, to be raised to the fourth intercostal space and carried to the vertical line of the nipple, or, possibly beyond this line. This change, taken in connection with other signs, is highly significant of pericardial effusion. But it must be recollected that causes other than pericardial effusion alter, in the same way, the point of apex-beat, for example, tympanitic distension of the stomach. An abundant accumulation of liquid suppresses all impulse. When the beat is found to disappear, after having been previously felt even more forcibly than in health, if the presence of liquid have been already determined, an increased amount of accumulation is to be inferred; afterward, as absorption of the liquid goes on, the heart's impulse becomes again appreciable, at first elevated above its normal position, and it may, or it may not, subsequently fall to the point where it was felt prior to the effusion of liquid.

It is to be borne in mind that various morbid conditions, other than pericardial effusion, may cause suppression of the apex-beat; such as pleuritic effusion, pulmonary emphysema, dilatation of the heart, fatty degeneration, etc. Excluding these, the following series of events occurring successively in connection with other signs denoting pericarditis, becomes highly diagnostic: increased force of the apex-beat, followed by diminished force, the point where it is felt raised and carried to the left, suppression of the beat, and its subsequent reappearance at, or somewhat above the point where it is felt in health.

A physical sign determined by palpation is a sensible vibration of the thoracic walls in the præcordial region, caused by friction of the pericardial surfaces roughened by an abundant deposit of dense lymph. The sensation communicated to the hand is analogous to that which accompanies a friction-sound due to the respiratory movements in some cases of pleuritis. This sign, first described by Stokes, is called the *pericardial friction-fremitus*. It is produced by the same physical conditions which give rise to friction-sound, and it is always accompanied by the latter. It occurs, however, only in a certain proportion of the cases in which a friction-sound is heard, and it rarely continues as long as the latter. It requires a greater degree of roughness of the pericardial surfaces than always obtains in pericarditis, and, also, a certain amount of vigor in the heart's movements. It occurs prior to the period of effusion, but very rarely after this period, the movements of the heart, at this stage of the disease, not being sufficiently strong to produce it. It is not incompatible with a small amount of liquid effusion, but it invariably ceases when the quantity of liquid is large. It is not to be confounded with the purring thrill which occurs in connection with valvular lesions. It is also to be discriminated from pleural tactile fremitus. The latter, being produced by the respiratory movements, ceases when respiration is suspended; while pericardial fremitus, being produced by the movements of the heart, is not arrested by holding the breath. The sensation of rubbing, or friction, distinguishes it from purring thrill. As a physical sign, the significance of tactile pericardial fremitus is neither more nor less than that of a rough friction-sound.

It is stated by Dr. Walshe that when the pericardial sac contains a certain quantity of liquid, the heart's impulse is sometimes felt after the systolic sound is perceived by the ear, a distinct interval separating these events which in health occur synchronously.

Signs furnished by inspection.

During the period of liquid effusion, if the pericardial sac be considerably distended, an unnatural prominence or arching of the præcordial region is sometimes apparent to the eye, especially if the subject be young. This sign of pericardial effusion was first pointed out by Louis. It is less frequent and marked in cases of acute pericarditis, than when the disease assumes a chronic form, with a very large accumulation of liquid. The effects of great distension of the pericardium, as determined by the different methods of exploration, will be noticed under the head of chronic pericarditis. Whenever the accumulation is sufficient to occasion an obvious projection of the præcordia, the intercostal spaces are, at the same time, widened and raised to the level of the ribs. These appearances are limited to a space on the chest corresponding to that which the distended pericardium occupies; and the prominence may present, indistinctly, an outline of the pyriform shape of the pericardial sac. Under these circumstances the impulse of the heart is rarely either seen or felt, a fact which serves to distinguish prominence of the præcordia produced by pericardial effusion, from that due to enlargement of the heart. In the latter case, one or more points of impulse are usually both seen and felt.

If the quantity of liquid be sufficient to produce visible enlargement of the præcordia, the respiratory movements on the left side are somewhat restrained. A disparity between the two sides, in this respect, may be obvious. The respiratory movements of the left side may also be restrained prior to the occurrence of effusion, in consequence of the pain felt during inspiration. Deficient respiratory motion is not, therefore, alone a sign that effusion has taken place. According to Dr. Walshe, if the quantity of effusion be moderate, the costal expansion of the left side may even be greater than in health, in compensation for some depression of the diaphragm.

Inspection is sometimes important as a means of determining the situation of the apex-beat of the heart, in cases in which this may be seen and not felt. Not infrequently the motion occasioned by the beat may be discovered by the eye, when it cannot be appreciated by the touch.

Undulatory movements in the intercostal spaces over the pericardium distended with liquid, are occasionally observed, due to

motion produced by the heart's action. This sign possesses small intrinsic value, *first*, because of its infrequency, and *second*, because it is not easy to distinguish it from the movements styled by Dr. Walshe *quasi undulatory*, caused by the motions of the heart itself when much dilated and in contact with the thoracic parietes over an enlarged area. The latter I have often observed, while it has not occurred to me to witness true undulation from liquid. Occurring in concurrence with other signs denoting unequivocally pericardial effusion, it has a positive significance.

If the quantity of liquid effusion have been sufficient to occasion a visible prominence of the præcordia, the removal of the liquid by absorption may be followed by an obvious præcordial depression. This is sometimes marked, and is important as one of the signs of ancient pericarditis.

Signs furnished by mensuration.

When the præcordia is rendered abnormally prominent by distension of the pericardial sac, during the period of effusion, this fact may be determined by mensuration. Diametrical measurement, with callipers, is best adapted to this object, for the same reasons as in cases of prominence due to cardiac enlargement. The normal deviations from equality of the two sides of the chest, as regards the results of diametrical measurement, are to be borne in mind.[1] The existence of undue præcordial prominence is determined by the eye sufficiently for practical purposes; but the callipers may be employed in confirmation of the evidence afforded by inspection. Owing to the rapidity with which, in certain cases, the liquid, on the one hand, accumulates, and, on the other hand, diminishes, præcordial prominence may be suddenly produced or increased, and as suddenly diminish or disappear. These variations may be ascertained with greater precision by mensuration than by the eye, and in recording cases which may be reported, it is more satisfactory to note the results of measurement, in addition to the appearances presented on inspection.

SUMMARY OF THE PHYSICAL SIGNS OF ACUTE PERICARDITIS.

Percussion.—Enlarged area of præcordial dulness; the extent of this area greater in a vertical than in a transverse direction; the

[1] *Vide* Chapter I. page 69.

shape of the area corresponding to the pyramidal form of the pericardial sac when distended; the dulness within this area, and the sense of resistance on percussion greater than over the præcordial region in health, or in cases of enlargement of the heart. These signs denote an abundant effusion within the pericardial sac. Moderate or small effusion denoted by increased width of the area of dulness at the lower and middle portions of the præcordial region. The increase of the area of dulness taking place within a few days or hours, and progressing rapidly; its extent varying on different days during the course of the disease. Dulness from the presence of liquid below the point of the apex-beat of the heart. Diminution of the area of dulness, with more or less rapidity, in the progress of the disease toward convalescence, and its final reduction to its normal limits when convalescence is established.

Auscultation.—A friction-sound developed, usually, soon after the commencement of the inflammation, depending, in general, on the exudation of lymph; rarely wanting during the period of the disease which precedes that of liquid effusion; frequently, not invariably, disappearing during the period of effusion; often returning after the absorption of liquid, and sometimes persisting after adhesion of the pericardial surfaces has taken place. Intensification of the heart-sounds at the commencement of the disease, or prior to liquid effusion; during the period of effusion, both sounds weakened, but especially the first sound; the element of impulsion in the first sound notably impaired or lost, and this sound, therefore, consisting of the valvular element alone, resembling the second sound as regards quality and duration; the sounds apparently distant, and the apparent distance greater when the patient is in some positions than in others. Cessation of respiratory murmur and vocal resonance, concurring with the results of percussion in determining the enlarged area of præcordial dulness dependent on distension of the pericardial sac.

Palpation.—Prior to the period of effusion, the cardiac impulse abnormally forcible, violent, extending over a larger space than in health, and sometimes tumultuous beating of the heart. After effusion, the point of apex-beat raised and carried to the left of its normal position. Suppression of the apex-beat, if the quantity of liquid be large. Return of the beat when the liquid diminishes. Vibration of the thoracic walls in the præcordia before, and some-

times after the period of effusion, constituting tactile friction-fremitus. Retardation of the apex-beat in some cases, after a certain amount of effusion, so that the first sound precedes it by a distinct interval.

Inspection.—Prominence or arching of the præcordial region in some cases during the period of effusion, if the pericardial sac be distended, observed chiefly in young subjects; the prominence presenting an indistinct outline of the pyriform shape of the pericardial sac. Restraint of the respiratory movements of the left side, if the quantity of liquid be large, and, also, prior to effusion, in some cases, from pain felt in the act of inspiration. Undulatory movements in the intercostal spaces over the pericardium distended with liquid, in a very small proportion of cases. Depression of the præcordial region in some cases, after the absorption of liquid.

Mensuration.—Prominence of the præcordia, in some cases, produced by liquid accumulation in the pericardial sac, determined by callipers. Sudden development or increase of prominence, and its sudden or rapid disappearance.

Diagnosis of Acute Pericarditis.

The diagnosis of pericarditis, until within a few years, was confessedly difficult in all cases, and often impossible. So long as the discrimination rested mainly on symptoms, it could rarely be made with positiveness. Laennec candidly acknowledged that the disease was not to be recognized, but its existence only conjectured. It was seen, while passing in review the symptomatic phenomena, that none of these are distinctive. As regards symptoms pointing to the heart, the disease is not infrequently absolutely latent. Moreover, in a certain proportion of cases, it is associated with other affections which, as it were, drown its manifestations. The disease now, as heretofore, is very rarely, if ever, ascertained to exist with certainty by those who rely in diagnosis on symptoms alone. And since physical exploration is still neglected to a great extent, or imperfectly understood, pericarditis is habitually overlooked by a large proportion of medical practitioners. With the aid of physical signs, the diagnosis may generally be made with ease and confidence. These have been sufficiently considered, but

there are certain sources of inadvertency and embarrassment which, in order to be avoided, should be clearly understood and impressed upon the mind.

The disease is liable to pass undetected, because its existence is not suspected, and attention is not directed to the condition of the heart. It is important to bear in mind the pathological relations of the disease, in order to be prepared to expect it, and to be on the watch for the earliest evidence of its development. During the progress of acute rheumatism, the præcordial region should be daily explored with reference to the signs of pericarditis, as well as those of endocarditis. By so doing, a friction-sound may sometimes be discovered when the patient makes no complaint of pain or other symptoms denoting that the pericardial structure has become involved. So, when a patient is known to be affected with Bright's disease, or albuminuria, the fact that inflammation of the pericardium, as well as other serous structures, is apt to be developed, is not to be forgotten, and examinations of the chest should not be neglected. In the eruptive and continued fevers, attention should be directed to the heart, and, indeed, it is well to adopt the practical rule enforced by Hope, which has been already mentioned, viz., to employ at least palpation habitually in all cases of disease.

The coexistence of pleuritis, or pleuro-pneumonia, is liable to lead the practitioner to overlook pericarditis, *first*, because, having ascertained the existence of these affections, he may attribute all the symptoms to them, and not carry his inquiries farther; and, *second*, because these affections obscure the symptoms, and, to some extent, the signs of pericardial inflammation. The diagnosis of the latter is, in fact, sometimes difficult under these circumstances. The heart should be interrogated as far as possible. A friction-sound may be discovered even when the organ is displaced to the right of the sternum. Symptoms referable to respiration and the circulation, out of proportion to the pulmonary affection, should excite strong suspicion of cardiac disease. Liquid effusion in the pericardium when, at the same time, there is an abundant effusion in the left pleural sac, is by no means easily determined; the physical signs may not be available for a positive diagnosis; but, with due attention, the pericardial accumulation may sometimes be disconnected from the pleural by prominence of the præcordia, the vertical extent of præcordial dulness, etc. An abundant effusion into the right pleural sac does not interfere materially with the

signs of pericardial effusion, and the diagnosis of the latter may be made with positiveness. The occasional occurrence of a cardiac pleural friction-sound is to be recollected in connection with the subject of pericarditis associated with pleurisy seated in the left side of the chest. This sign is a source of fallacy against which there is no absolute protection, and this fact should lead the practitioner not to commit his mind too unqualifiedly to a diagnosis based exclusively on the existence of a friction-sound.

I have known acute pericarditis, disconnected from any other thoracic affection, to be considered and treated throughout the disease as pleurisy; but the diagnosis was based on symptoms alone. A tolerable knowledge of physical exploration enables the diagnostician to exclude, on the one hand, and, on the other hand, ascertain the existence of pleurisy and pneumonia. If the examination of the chest be limited to the anterior surface, the physical signs of liquid within the pericardial sac might be attributed to pleural effusion in the left side. A proper examination of the whole chest obviates liability to this error. The signs of effusion are limited to the anterior surface. Percussion and auscultation show the presence of lung in the lower posterior portion of the chest; and it is precisely in the latter situation that the signs of pleuritic effusion are first manifested and most marked. This error is excusable on no other ground than inability to employ the means of arriving at a correct diagnosis. The occasional occurrence of symptoms referable to the brain and spinal cord, in connection with pericarditis, is to be borne in mind. These symptoms may mask completely those pertaining to the cardiac affection, simulating various affections of the nervous system, viz., mania, apoplexy, tetanus, etc. The peculiar characters which serve to distinguish these cases are to be kept in view, and careful attention directed to the heart in all instances in which these affections appear to be present.

Pericardial effusion, occurring without inflammation, has not yet been referred to. Dropsy of the pericardium, or hydro-pericardium, rarely occurs to an extent sufficient to occasion great distension of the sac. It occurs very rarely, if ever, except in conjunction with effusion into other serous cavities, and the areolar tissue, constituting general dropsy. The effusion into the pleural cavities is proportionately greater than into the pericardial cavity. The physical signs of a certain quantity of purely serous or dropsical effusion are, of course, the same as when the effusion is combined with

lymph, or, in other words, inflammatory. The pericardial sac, in proportion to its distension, occupies the same space, enlarges the area of dulness to the same extent, the latter presenting the same pyriform shape, etc., in the two cases. The discrimination, however, rarely offers much real difficulty. Liquid effusion in pericarditis is generally preceded and accompanied by more or less of the symptoms pointing to the latter affection, such as pain, tenderness, febrile movement, etc. It is preceded almost invariably, and not infrequently accompanied by, a cardiac friction-sound. If pleuritic effusion be also present, as determined by physical signs, it is not hydro-thorax, but due to coexisting inflammation of the pleura; and the inflammatory pleuritic effusion is generally limited to one side. On the other hand, hydro-pericardium is an element of general dropsy; œdema or anasarca, ascites, and hydro-thorax, are at the same time present. A friction-sound is never developed. In the vast majority of cases, the patient is affected with either Bright's disease or organic disease of heart, and both affections may be united. With due attention to these differential points, the two kinds of effusion, viz: dropsical and inflammatory, need not be confounded.

In leaving the subject of diagnosis, the great importance of becoming familiar and practically conversant with the physical signs is to be impressed. With this knowledge, the practitioner will rarely be long at a loss in determining whether acute pericarditis be, or be not present.

Prognosis in Acute Pericarditis.

Acute pericarditis is never a trivial, and is often a formidable affection. The fatality, however, is due, not so much to the disease itself, as to the condition of the system, the pathological relations of the disease, and coexisting affections. Cases of idiopathic pericarditis are so rare that that statistical data for determining the rate of mortality are, as yet, wanting. Of 106 cases of pericarditis variously complicated, which were analyzed by Louis, 36 died. The reports of different observers, as regards the proportion of fatal cases in their own experience, differ considerably; and this would be expected, in view of the great variation in the tendency to a fatal result, according to the different circumstances under which the disease is developed. Dr. Hope, whose opportunities for

observation must have been extensive, makes the remarkable statement that, in ten years, he had not lost a patient with acute pericarditis, ascribing this success to the treatment pursued. Yet, according to the observations of others, the disease is almost invariably fatal, when developed in certain pathological connections.

In the great majority of cases, pericarditis occurs in connection with either acute rheumatism or renal disease. Occurring in connection with rheumatism, it rarely proves fatal. In 84 cases reported by Latham, McLeod, and Bouillaud, there were but 8 deaths; and, in more or less of these fatal cases, endocarditis coexisted. Rheumatic pericarditis, thus, may be expected to end in recovery, or, at least, not to terminate fatally as an acute affection. It is otherwise when the affection is developed in connection with Bright's disease; a fatal result occurs almost invariably. Death takes place in a large proportion of the cases in which it is associated with pleurisy or pneumonia; but, in many, if not most of these cases, it is probable that the kidneys are the seat of disease. The proportion of fatal cases of pericarditis developed in connection with the eruptive and continued fevers, pyæmia, etc., must be immensely large; but statistical data are wanting to determine the ratio with any approximation to exactness. The disease is generally fatal when associated with marked disorder of the nervous system, giving rise to mania, tetanus, chorea, etc.

As regards the fatality in my own experience, the following are the results of an analysis of 19 recorded cases, with reference to this subject: Of the 19 cases, death occurred in 12, and in 7 the disease ended in recovery. Of the 12 fatal cases, renal disease, with albuminuria, was ascertained to exist in 3; pleuritis coexisted in 5; pneumonitis in 2; tuberculosis of the lungs in 3; maniacal delirium in 2; and in 1 case no important complication was ascertained. In the case last mentioned, the disease did not present any alarming symptoms, but death occurred suddenly, apparently from syncope, while the patient was at stool. The pericardial sac contained a pint of turbid serum. The heart presented two patches of lymph, one of the size of a dollar, and the other of the size of a shilling piece. The pulmonary organs were free from recent disease, but old pleuritic adhesions existed on both sides, and they were universal on the right side. The chest was alone examined. Of the 7 cases ending in recovery, in 4 the disease was developed in connection with rheumatism; in 1 case it was apparently idiopathic, and was

associated with maniacal delirium; in 1 case pneumonia coexisted; and in 1 case it followed albuminuria, succeeding scarlatina.[1]

The duration of the disease is variable. It may prove rapidly fatal. In a case reported by Andral, death occurred in twenty-seven hours; but it is extremely rare that it runs with this rapidity to a fatal issue. It continues, usually, from one to two weeks. If it do not prove fatal within this period, it ends either in recovery or in the chronic form of the disease. The latter will presently be considered under a distinct head.

The termination, in favorable cases, is usually in more or less adhesion of the pericardial surfaces. It may fairly be doubted whether the exuded products are ever completely removed by absorption, leaving the surfaces of the membrane unattached, and presenting no traces of the disease. Some, however, have contended that this complete resolution occasionally takes place. As a rule, certainly, permanent effects are left here, as after recovery from inflammation affecting other serous structures, consisting of adhesions by means of newly organized tissue which becomes more and more firm with age. Adhesion may take place over the entire surfaces of the membrane, the pericardial sac being obliterated, like that of the tunica vaginalis after the radical cure of hydrocele. In fact this appears to be the rule. Of 70 cases of pericardial adhesions analyzed by Louis, they were general in 60, and partial in 10; and of 86 cases of old adhesions analyzed by Dr. Chambers, 51 were universal, 4 nearly so, and 29 partial.[2] The subject of pericardial adhesions, with reference to their remote effects upon the heart, and the diagnosis, will be noticed separately, after chronic pericarditis has been considered. The most favorable termination of acute pericarditis, next to complete resolution (the occurrence of which is doubtful), is the formation of circumscribed white patches, consisting of thin layers of dense lymph, firmly agglutinated to the membrane, becoming nearly as smooth and polished as the membrane itself. These white patches (*maculæ albidæ*), as an effect of circumscribed or partial pericarditis, have been already noticed. It is possible that patches similar to these are, in some cases, the

[1] It is proper to state that the foregoing collection of cases does not include all that have fallen under my observation, but only those of which I find notes among my clinical records. It probably embraces the greater proportion of fatal cases which I have observed, while, of a considerable number of cases of rheumatic pericarditis not ending fatally, I have not preserved notes.

[2] Decennium Pathologicum. *Vide* Bellingham, op. cit., Part II. p. 309.

only permanent effects of acute, general pericarditis; but this must be considered as doubtful.

The mode of dying, in cases of acute pericarditis, is not uniform. When life is destroyed by the disease *per se*, the result is immediately due either to sudden syncope or gradual asthenia. The arrest of the circulation is the immediate cause of death; and this is owing to paralysis of the heart, usually from the combined influence of the mechanical pressure of liquid effusion, and the proximity of the inflamed membrane to the muscular fibres of the organ. In the latter respect, the influence is analogous to that of inflammation of the peritoneum on the muscular coat of the intestines. It is important to keep in view this twofold influence in producing a tendency to a fatal result, since it should govern, to a considerable extent, the indications for treatment. But in the great majority of fatal cases of pericarditis, concomitant affections contribute, in no small degree, to the fatal result, and the mode of dying will, in a measure, be determined by these. For example, when the disease is associated with pleurisy or pneumonia, asphyxia is involved as an immediate cause of death. Again, when developed in the course of Bright's disease, the powers of the system being exhausted by the latter, the tendency to death may be by slow asthenia; or coma may be induced as an effect of uræmia, not of the cardiac affection. An example of the latter has fallen under my observation. Coma precedes death in the cases in which pericarditis simulates various affections of the brain and spinal cord. A liability to sudden death from syncope, during the period of effusion, must not be overlooked. This may occur after some unusual muscular exertion, or a sudden change from a horizontal to a vertical position. An instance in which death occurred suddenly and quite unexpectedly, in a case under my observation, has been referred to, fatal syncope being induced by getting out of bed and going to stool. The inflammation in this case, as shown by the appearances after death, as well as the symptoms during life, was not intense, and the pericardial sac did not contain more than a pint of liquid effusion.

Treatment of Acute Pericarditis.

In the treatment of most acute inflammations, the general symptoms, the condition of the system, the pathological relations of the

disease, etc., are more immediately involved in therapeutical indications, than the local processes which constitute the inflammatory affection. The treatment of acute pericarditis does not form an exception to the rule embodied in this statement. Acute pericarditis is developed under circumstances so widely different in different cases, that, assuming the local characters of the inflammation to be identical, the indications for treatment are by no means uniform. Measures useful in some cases are pernicious in other cases. Methods of management diametrically opposite, are indicated by different circumstances connected with the disease. It follows that the treatment cannot be reduced to a fixed formula applicable to all cases. Here, as in other affections, the significant saying of Chomel is pertinent, viz., the disease is not to be treated so much as the patient affected with the disease. In the majority of cases, pericarditis occurs in connection with acute rheumatism. Exclusive of its occurrence in this pathological connection, it is most apt to become developed in the course of albuminuria or Bright's disease. The local inflammation, in all essential points, so far as these are appreciable, may be the same in these two classes of cases, but in other respects, the difference is very great. Rheumatic pericarditis involves small immediate danger to life, although its remote evils may be serious. In renal pericarditis (if this expression may be used) the immediate danger to life is imminent. The chances of recovery in the latter are less than the chances of death in the former. Both forms of the disease are dependent on special diathetic or constitutional conditions which are essentially dissimilar. The active rheumatic diathesis is acute, transient in duration, affecting the young; the uræmic condition is incident to a chronic, persistent affection, and occurs at a later period in life. The ability to bear up under any grave local disease, and to support potent remedial agencies, is as different as are the pathological relations of the disease in these two forms. In addition to these, a variety of modifying circumstances are present in different cases of pericarditis, not peculiar to this disease, but affecting the symptoms, the powers of the system, etc., so as to influence, in various and opposite modes, the leading objects of treatment. It is not to be inferred from these remarks that, in the treatment of pericarditis, great importance does not belong to the local morbid conditions. It is by means of the vital and mechanical effects of the latter that the disease proves destructive to life. The objects of treatment relate to the local morbid conditions; but in promoting these

objects, the indications are in a great measure derived from the circumstances just referred to.

It is obvious that the first and most important object of treatment, were it attainable, would be the arrest of the inflammation. But we are not warranted in assuming this as an object to be effected by therapeutical measures. With our present knowledge, we cannot say that certain methods of management will cut short the inflammatory processes here, more than in other situations. Potent means adopted for that end, not only prove ineffectual, but involve the risk of doing harm rather than good. To abridge the duration of the inflammation is an object, the importance of which is sufficiently obvious. How far this object is attainable, must be considered as doubtful, but we are perhaps justified in regarding it as an end to which therapeutical measures are to be directed. To endeavor to diminish the intensity of the inflammation, is a legitimate object of treatment. The products of inflammation, solid and liquid, in this situation, involve serious evils and danger. It is an object of treatment to endeavor to lessen these, and to promote their removal. To aid in maintaining the vigor of the heart under the effects of the disease, is an important end to be kept in view in the treatment. I shall proceed to notice the more important of the therapeutical measures which are supposed to promote the objects or ends of treatment in acute pericarditis. These may be arranged under the following heads: Bloodletting, mercurialization, sedatives, revulsives or counter-irritants, opium, stimulants, and eliminatives.

Bloodletting.—The two authors whose labors have contributed most to the recent progress made in the knowledge of diseases of the heart, viz., Bouillaud and Hope, both advocate strongly the importance of bloodletting in cases of acute pericarditis. Bouillaud employs, in this disease, his method of copious bleedings, repeated once or oftener daily for four or five successive days (*coup sur coup*), with which medical readers are familiar. Hope's method differs from that of Bouillaud in the employment of bloodletting only at the outset, and carrying it to the extent of producing a prompt and decided impression on the heart's action. Each advocates the detraction of blood both by venesection, and, locally, by leeching or cupping. Writers of a more recent date (Stokes, Todd, and others) distrust the efficacy of this remedy, and attribute to it in many cases unfavorable effects. Without entering into a discussion of

the subject of bloodletting, which would here be out of place, it is sufficient to say that, with certain qualifications, the general principles which should regulate the employment of this potent remedy in other acute inflammations, are applicable to the treatment of acute pericarditis. Its indiscriminate use in this, as in other inflammations, cannot but be productive of much harm, whereas, judiciously employed, it may not infrequently do good. The practical questions are, under what circumstances is bloodletting indicated, and what are the contra-indications to its use? A person in fair health and vigor, attacked with acute pericarditis, as an idiopathic or a rheumatic affection, is a proper subject for bloodletting at the onset of the disease. Resorted to under these circumstances, it will not cut short the disease, and perhaps not abridge its duration; but it may contribute to diminish the intensity of the inflammation, and thus, without risk of injury, not only afford immediate relief, but lessen the evils and the danger, proximate and remote, which are involved in the disease. The amount of blood to be detracted, must be determined by the constitution, habits, etc., of the patient, the symptoms referable to the circulation, and the immediate effects upon the vascular system. Whether the bloodletting shall be general or local, or both, is to be determined mainly by the quantity of blood which it is deemed desirable to withdraw, and the comparative convenience of venesection and cupping or leeching. It is difficult to conceive of any important difference between these different methods, as regards their effect on the disease, except so far as concerns the rapidity with which the blood is removed, and the amount detracted. The benefit derived from bloodletting will be evidenced by relief of pain, greater freedom of breathing, diminished force and greater regularity of the heart's action. These, then, are the circumstances which may indicate bloodletting, viz., the disease idiopathic, or rheumatic; occurring in a patient previously healthy and tolerably vigorous; the inflammation recently developed, or, in other words, the disease being in its first period, and, to these is to be added, a certain degree of intensity or acuteness of the inflammation, as manifested by pain, development of the pulse, etc. The indications based on these circumstances are present in a certain proportion of cases of pericarditis. The contra-indications, however, are present in a larger proportion of cases. Pericarditis occurring in connection with Bright's disease, rarely, if ever, calls for bloodletting. The anæmic condition incident to that disease, constitutes a contra-indication. Anæmia from other causes, weak-

ness or deterioration of the constitution from previous or coexisting disease, habits of intemperance, etc., are contra-indicating circumstances. Bloodletting should not be practised after much liquid effusion has taken place; it is contra-indicated by the risk of unduly weakening the heart under these circumstances. It is not indicated when the symptoms denote only moderate acuteness or intensity of the inflammation.

In conclusion, as regards bloodletting, the mischief occasioned by its injudicious employment may greatly exceed the benefit ever to be expected from its judicious use. This only shows the great importance of discrimination; and the remark is alike applicable to this remedy in the treatment of other inflammations. But the injudicious detraction of blood in pericarditis involves a peculiar source of danger. It has been seen that this disease destroys life by compression and paralysis of the heart. Any remedy which tends directly to weaken this organ, when weakness is the morbid condition to be most apprehended, can hardly fail, in proportion to the effect produced, to influence unfavorably the progress of the disease. This consideration cannot be too strongly impressed. Here, as in other affections, the attention of the practitioner must not be directed exclusively to the good which it is hoped may be effected by a potent remedy. The risk of harm is to be carefully weighed, and, with reference to the latter, the mode in which the disease tends to a fatal result is especially to be considered.

Mercurialization.—Mercury, given with the view of producing its special effects, or mercurialization, is regarded, especially by most British writers, as highly essential in the treatment of acute pericarditis. It is supposed to exert a favorable influence on the progress of the disease by lessening the exudation of lymph and promoting the resorption of the inflammatory products. With reference more particularly to the first of these ends, it is deemed important to induce mercurialization as early in the disease as possible. Calomel may be given for this purpose, either in fractional doses, repeated at short intervals, or in large doses, combined with sufficient opium to prevent its purgative action; and, in order to effect this object as speedily as possible, some writers advise, in addition, inunction with mercurial ointment, or the mercurial vapor-bath. Bouillaud, and most French writers, on the other hand, appear to attach little importance, or none whatever, to the special effects of mercury in this disease. It is claimed by some British

writers that the disease is managed much more successfully in Great Britain than in France, and that this greater success is owing, in a great measure, to the free use of mercury. The testimony, however, of some British observers, on this score, is unfavorable to the efficacy of the remedy. Dr. Taylor, for example, found that in a considerable number of cases in which salivation was induced, a speedy abatement of the disease did not take place, and in several instances it was increased in extent and intensity.[1] With reference to this and other therapeutical questions, satisfactory statistical data are as yet wanting. Nor is much to be expected from statistics in determining the value of remedial agencies in a disease which varies so greatly in severity and danger, according to its pathological relations and other circumstances, and which is not sufficiently common for a large number of cases to fall under the observation of any one practitioner. Moreover, here, as with regard to many other acute affections, the natural tendency of the disease, uninfluenced by remedial measures, is not fully ascertained, and it must be long ere the data for this knowledge can be acquired.

Mercury is advocated in pericarditis on precisely the same grounds as in other inflammations—for example, pleurisy or pneumonia. But confidence in the utility of the remedy in the latter affections has of late years greatly diminished, and at the present moment, many judicious practitioners do not deem its special effects in the treatment of these affections either demanded or desirable. Distrust of the supposed influence of mercurialization upon the process of exudation and the removal of morbid products, is evidently gaining ground. Even iritis, the affection which has been heretofore regarded as affording convincing ocular proof of the power of mercury in effecting the removal of lymph, has been shown by Dr. Williams, of Boston, to progress quite as favorably, if not more so, when this remedy is withheld.[2] Moreover, as regards the influence of mercurialization on pericarditis, the fact that, in the course of acute rheumatism, the disease has been repeatedly observed to become developed during salivation, militates against the applicability of mercury as a remedy. Dr. Fuller gives several instances

[1] Bellingham, op. cit., pt. ii. p. 326.

[2] On the Treatment of Iritis without Mercury. By Henry W. Williams, M. D. Reprinted from the *Boston Medical and Surgical Journal*, 1856. The conclusions in this paper are based on the results of the treatment of sixty-four cases of iritis without mercury. These results are of great interest and value in their bearing on the non-mercurial treatment of inflammations generally.

in illustration of this fact, and examples have fallen under my own observation. Confessing doubt concerning the propriety of mercurializing patients affected with pericarditis, I am not prepared to deny, *in toto*, the importance of this method of treatment, inasmuch as the inconvenience and evils incident to moderate salivation, in many cases of this disease, are hardly deserving consideration, provided the remedy be entitled to a tithe of the value which is claimed for it.

Assuming a certain amount of efficacy in behalf of mercurialization, it is not adapted to all cases. It is allowable chiefly in cases of idiopathic and rheumatic pericarditis. It is contra-indicated by the coexistence of Bright's disease, and is not appropriate whenever pericardial inflammation is developed in connection with anæmia or a broken constitution. The importance of this discrimination is conceded by those who advocate strongly the importance of the remedy.

Sedatives.—Under this head, I refer to certain drugs which depress the powers of the system, and particularly the action of the heart, such as antimony, digitalis, the veratrum viride, etc. The excitement and disorder of the circulation often incident to the first period of pericarditis might perhaps suggest the employment of remedies of this class. In general, they are not appropriate. Employed during the period of effusion, especially if the quantity of liquid be large, they are dangerous remedies if carried to the extent of weakening or retarding considerably the movements of the heart. They are only admissible before and after the period of effusion, and are rarely indicated at any time during the career of the disease.

Revulsives or Counter-irritants.—These are useful for the same reasons, and probably to the same extent, as in the treatment of inflammation affecting analogous structures, for example, pleurisy; and the general principles which should govern their employment are the same. During the early and most acute period of the disease, blisters and other active modes of counter-irritation are inadmissible. Revulsive measures, such as sinapisms, fomentations, and foot-baths, are, to a certain extent, useful during this period. Vesication over the præcordia when the pericardial sac is distended, and the intensity of the inflammation has abated, probably hastens absorption, as it apparently does in cases of pleurisy with effusion.

But there is this objection to the application of blisters directly over the heart: they interrupt daily physical exploration in order to determine the quantity of liquid effusion, etc. After the absorption of the liquid, it is possible that moderate counter-irritation, maintained by small blisters, an issue, or pustulation with croton oil, may serve to bring the inflammation speedily and completely to an end, preventing a termination in the chronic form of the disease. The pathological relations of the pericarditis and the state of the system are to be taken into account in deciding on the propriety of counter-irritation and the extent to which it is to be carried. The practical question is, Will the probability of its superseding the disease warrant the risk of adding to the irritation and exhaustion of the disease if revulsion be not effected? This is a therapeutical problem which the practitioner cannot be expected to determine positively in individual cases, except by employing experimentally counter-irritant measures.

Opium.—The utility of opium is generally admitted. But there are grounds for the belief that the value of this remedy in the treatment of pericarditis has not been hitherto, and is not now, generally appreciated. In analogous local inflammations—peritonitis, pleuritis, pneumonia, and perhaps may be added meningitis—opium exerts often a remarkable influence, not merely as a palliative, but as a remedial agent. It relieves pain and quiets functional excitement; but, more than this, it appears to control, to a considerable extent, the processes of inflammation, abating its intensity, abridging its duration, and contributing to a favorable termination. To secure its full potency, it must hold a leading, not a subordinate place in the management; and clinical experience shows that in these affections there is often a remarkable tolerance of the remedy, so that, to produce a proper effect, large doses are frequently requisite. Analogy would lead to the expectation of a similar remedial power in cases of acute pericarditis. It remains to accumulate a sufficient number of cases in the treatment of which reliance has been chiefly placed on the free use of opium, in order to confirm the correctness of this inferential reasoning. It will be understood that these remarks have reference to opium as a prominent remedy in the treatment of pericarditis. As a subsidiary remedy, its value is already sufficiently attested by experience.

The use of opium has this advantage over other measures, viz.,

it is not positively contra-indicated by any of the various and opposite circumstances associated with the disease. If it be appropriate in rheumatic pericarditis, it is equally so when pericardial inflammation is developed in the course of Bright's disease, or in other pathological relations. If it be not efficacious, it is not mischievous, except so far as it may supplant other measures and involve loss of time. It admits of being employed tentatively without incurring much, if any, risk of doing harm, even by delay. Its potency for evil is not proportionate to its potency for good, and this cannot be said of most potent remedial agencies.

The periods of the disease most favorable for the beneficial influence of opium are the period anterior and that subsequent to liquid effusion. It is, however, by no means certain that the remedy affects unfavorably absorption of the effused liquid. I have known an abundant pleural effusion, in connection with pleuro-pneumonia, to disappear rapidly when no other remedy was employed.

Stimulants.—In all inflammatory affections, diffusible or alcoholic stimulants form an essential part of the treatment, whenever measures to sustain the power of the system, and obviate the tendency to death by asthenia, are indicated. Pericarditis is by no means exempt from these indications; on the contrary, since the immediate danger from the disease chiefly arises from weakness of the heart, sustaining measures would seem to be called for earlier and more imperatively than in most local inflammations. As remarked by Dr. Stokes, little is said by authors on the use of stimulants in pericarditis. This distinguished author adds: "I am convinced that cases are often lost from want of stimulation at the proper time; and it is certain that, in every case of dangerous pericarditis, after the first violence of the disease has been subdued, we should be anxiously on the watch for the moment when the weakened heart requires to be supported and invigorated." In the treatment of inflammatory affections generally, timidity in the use of stimulants is apt to proceed from the attention of the practitioner being too exclusively directed to the local morbid processes, the state of the system being overlooked, or not sufficiently regarded. Measures designed to abate the intensity of inflammation, and to control its processes, pertain, for the most part, to an early period in the disease. After a certain time, all the immediate local results of inflammation, which may be expected to occur, have already taken place,

and the general object of treatment, then, is to maintain the forces of life through the processes of restoration. The physician, under these circumstances, is to regard the patient more than the disease, in looking for therapeutical indications. Alcoholic stimulants, given to support the flagging powers of life, do not excite, as in health, but, associated with nutriment, they sustain the vital forces, keeping the patient alive, in some cases in which death from asthenia is threatened, until the period of danger is passed. They are to be given with a freedom proportionate to the indications and the apparent effects. These general views, as applied to local inflammations indiscriminately, seem to me to possess very great practical importance. With regard to pericarditis, it is only necessary to add that they are at least as applicable to this as to any other inflammatory affection.

Eliminatives.—The propriety of eliminative remedies in pericarditis rests on the pathological relations of the disease in certain cases. As developed in connection with rheumatism and Bright's disease, the pathology is supposed to involve a *materies morbi*, the removal of which from the system may, perhaps, be promoted by remedies employed for that purpose. Thus, in rheumatic pericarditis, the question arises whether those remedies supposed to be efficacious as eliminative agents in rheumatism, may not diminish or expel a cause which serves to perpetuate the cardiac inflammation. It is obvious that remedies of this class, if they possess any efficacy, are more indicated as prophylactic than curative agents; and, with reference to the prevention of pericarditis in rheumatism, clinical observation shows that cardiac inflammation may become developed under any of the various methods of treatment which are pursued in the latter affection. Without discussing this important practical subject, I will simply say that we are hardly warranted in asserting that any one or more remedies can be relied upon to prevent the development of pericarditis in rheumatism; but, on the other hand, it is by no means improbable that certain measures may exert an influence more or less prophylactic. Of the remedies which have been supposed to possess a controlling power over acute rheumatism, by means of their eliminative effects, the most prominent are various diuretics, alkalies, and colchicum. It would be out of place to consider the relative merits of these, and it suffices to say, in so far as each or all may possess the efficacy claimed for them, it is not unreasonable to conjecture that they are

useful after pericarditis has been developed. Our knowledge with respect to this point, however, is not sufficiently precise to render it judicious to trust to eliminative measures in rheumatic pericarditis, to the exclusion or depreciation of other therapeutical means of more direct application in the treatment of the disease.

These remarks will apply, measurably, to pericarditis as developed in connection with Bright's disease. The morbid material here is supposed to be urea, or the products of its decomposition in the blood. The remedies supposed to act as eliminatives are diuretics, cathartics, and sudorifics. As regards the employment of these remedies with a view to elimination, in pericarditis, it is important to remark that they are contra-indicated, not only when they come into opposition to other measures of greater importance, but when, irrespective of their eliminative operation, they are likely to prove hurtful. This remark applies more particularly to active purgative remedies. The probability of good being effected by means of elimination is not sufficient to warrant taking the risk of doing injury. Remedies to act on the kidneys and skin may be given more safely. As a sudorific, Dr. Walshe advocates especially the hot air or vapor bath, which has these advantages over the warm bath, viz., it may be taken with the head moderately low, and without exertion on the part of the patient.

Having noticed the more important of the therapeutical measures embraced in the treatment of acute pericarditis, the practical points which have been presented may be recapitulated, and others added, in considering briefly the indications which belong, respectively, to the successive periods of the disease, viz., prior to, during, and after liquid effusion.

Treatment prior to liquid effusion.

The chief objects of treatment in this period are, abatement of the intensity of the inflammation; limitation of the products of inflammation (serum and lymph), and, perhaps, by effecting these objects, shortening the duration of the disease. The means which may be employed for these ends are, bloodletting, mercurialization, opium, and eliminatives.

Bloodletting, as a rule, is appropriate only in cases of idiopathic, and in certain cases of rheumatic pericarditis. It is contraindicated by coexisting Bright's disease, anæmia, feebleness, or a broken constitution. It is rarely, if ever, indicated when pericarditis occurs

in connection with the eruptive or continued fevers, pyæmia, etc It should be employed only when the inflammation has a certain degree of intensity, or shown by febrile movement and a firm pulse. The repetitions of bloodletting, and the amount of blood withdrawn, are to be determined by the symptoms and the effect, bearing in mind the danger of weakening too much the heart by this measure. It is to be employed only when physical exploration of the chest affords evidence that an abundant effusion of liquid has not yet taken place.

Mercurialization is appropriate only in cases of either idiopathic or rheumatic pericarditis, and when the constitution of the patient is not greatly impaired from any cause. Anæmia constitutes a contra-indication. If this measure be employed, the system should be brought rapidly under the effects of mercury, but it is never necessary to induce severe ptyalism; on the contrary, this is to be avoided, if possible. Patients with this disease are often mercurialized with difficulty. When this is found to be the case, it is better to relinquish the attempt, than to introduce a quantity of the remedy into the system, which may, in the end, lead to excessive effects.

Opium is safer and more reliable than either bloodletting or mercurialization. It may be employed in the cases in which these measures are contra-indicated; and it may be employed in conjunction with them. It should be given in doses sufficient to relieve pain and tranquillize the circulation. The doses required to produce a sufficient effect will sometimes be large, owing to the tolerance of the remedy in this disease.

Eliminatives are appropriate, with proper restrictions, in cases in which the disease is dependent on rheumatism or uræmia. These remedies, however, are subordinate to those which have a more direct influence on the disease.

Treatment during liquid effusion.

The chief objects of treatment in this period are, to prevent the progressive accumulation of liquid; to promote its resorption; to invigorate the heart and obviate danger from paralysis and compression of the organ. The means for these ends are, mercury, opium, counter-irritation, stimulants, nutritious diet, and to these may be added, diuretics and hydragogue cathartics.

The effusion of liquid does not contra-indicate mercurialization,

if the precautions already referred to, be observed. Limitation of the products of inflammation is still an object of treatment; and one of the grounds on which mercury is advocated, is its influence in promoting resorption of these products. Still greater care, however, is to be observed in employing this measure after, than before the disease has advanced to the second period.

Indications for opium may be present in the second, as well as in the first period, but not to the same extent. To relieve pain and quiet irritation, are still objects of treatment, and this remedy is indicated in doses sufficient to effect these objects.

Vesication upon or near the præcordia, which is not admissible in the first period, is now useful in promoting resorption, and also by way of revulsion. In order not to interfere with physical examinations of the chest, by means of which the progress of the disease, from day to day, is ascertained, it is preferable to apply blisters in the neighborhood of the præcordia, and not directly over the heart. The employment of a series of blisters applied in different situations, allowing the blistered surface to heal as quickly as possible, is best suited to promote absorption, whilst perpetuated blisters are, perhaps, most likely to act as revulsives.

Diffusible stimulants are indicated, in the second period, by weakness of the heart. They are indicated in proportion as the heart becomes weakened by paralysis and compression. Weakness of the heart is manifested by the pulse and other symptoms; but more distinctly by physical signs. Feebleness or suppression of the apex-beat, diminished intensity of the heart-sounds, more especially of the first sound, and extinction of the latter, call imperatively for measures to invigorate the heart. Alcoholic stimulants, in the form of either wine or spirits, are the most efficient means for this end. They should be given as freely as they are found to be well borne, the criterion being, not a certain quantity, but a certain effect. The desired effect is increased strength, with diminished frequency, of the pulse. The physical signs also afford a guide in regulating the quantity of stimulants. The reappearance or increased force of the apex-beat, and an approximative return of the sounds of the heart to their normal relative intensity—in other words, improvement in the first sound more particularly—denote the beneficial influence of stimulation.

In conjunction with stimulants, a nutritious diet is indicated. Whenever stimulants are useful, the diet cannot be too nutritious. Animal essences or tender meat itself, constitute the diet which is

most sustaining. But those articles of food are of course to be selected which are best adapted to the digestive powers in individual cases, and this is to be determined by experimental trials.

Diuretics and hydragogue cathartics may sometimes be employed with advantage, with a view to promote resorption of the effused liquid. But the employment of these remedies demands great circumspection. They are contra-indicated whenever stimulants and sustaining diet are required. This caution has reference more to hydragogue cathartics than to diuretics, but measurably to the latter.

Eliminative remedies may be continued, under proper restrictions, into the second period. They must not conflict with other measures upon which greater reliance is to be placed.

Aside from the indications just mentioned, purgative remedies are not advisable in the treatment of pericarditis, save to obviate discomfort attending constipation.

It will not be amiss to repeat the caution not to permit much exertion on the part of the patient, when the heart is compressed by an abundant accumulation of liquid effusion. Sudden and fatal syncope may be induced by the effort of rising from the bed to go to stool, as in an instance, referred to more than once, which came under my observation.

The importance of daily or frequent explorations of the chest, in order to determine the progressive diminution of the liquid effusion, and its final disappearance, has been repeatedly referred to, but it cannot be too strongly enforced.

Treatment after the absorption of liquid effusion.

The chief object of treatment in this period is to promote the complete disappearance of inflammation. The means for this end are, counter-irritation, tonic remedies, an invigorating diet and regimen.

It is highly probable that moderate, persisting counter-irritation near the præcordia tends, by way of revulsion, to expedite the final cessation of inflammation, and perhaps, in some cases, to prevent it from becoming chronic. The modes of counter-irritation suited to this object, which have been mentioned, are, perpetuated blisters, issues, and pustulation with croton oil. The counter-irritation should be so restricted in degree as not to produce constitutional disturbance, nor prove a source of exhaustion. Without this precaution, the evils would be likely to overbalance the revulsive effect.

In acute inflammations generally, the local processes of restoration go on more rapidly, the liability to relapse is less, and the final recovery is more complete, in proportion as the general powers of the system are invigorated. Hence, one great advantage in not employing unnecessarily, during the progress of the disease, debilitating measures of treatment. The less the patient is enfeebled, the more speedy and safe the convalescence. Hence, tonic remedies, a nutritious diet, cheerful relaxation of mind, and gentle exercise in the open air, are important so soon as convalescence is established. These are the means by which the body regains strength and vigor. They are applicable to pericarditis as well as to other inflammations. While undue exertion of body or mind, imprudent exposure, and excesses of all kinds, are, of course, to be avoided, the regimen and diet best calculated to restore or improve the general health will affect most favorably the condition of the organ recently inflamed.

In the treatment of pericarditis, associated affections, especially endocarditis, pleurisy, and pneumonia, will often claim attention. The measures to be directed to these affections need not be here considered. The treatment of the pericarditis, it is obvious, must be more or less modified under these circumstances. As a rule, they enforce greater circumspection in the use of debilitating remedies, and call for an earlier and more efficient employment of sustaining measures.

The danger in cases of pericarditis is much enhanced when the disease is complicated with notable disorder of the nervous system, giving rise to active delirium, convulsions, etc., symptoms which, it has been seen, often mask the cardiac affection. It is not easy to decide, with our present knowledge, as to the measures most likely to prove successful in this class of cases. In the case under my observation which ended in recovery, the treatment consisted of the free use of alcoholic stimulants, sustaining diet, and a blister upon the præcordia.

SUBACUTE AND CHRONIC PERICARDITIS.

Pericarditis may be subacute from the commencement. This explains the latency of the disease, as regards symptoms, in certain cases. In these cases, when the attention is first directed to the

chest, the pericardial sac may be found already largely distended. If death occur during the period of effusion, the liquid is found to be moderately or slightly turbid, a small quantity of lymph adhering, in circumscribed patches, to the pericardial surfaces. The disease is analogous to subacute pleurisy, as the latter is not infrequently presented in medical practice. In other instances, this variety of the disease succeeds the acute form. In acute pericarditis, if the inflammation do not disappear in the course of from two to three weeks, the disease may be considered as having become chronic.

The anatomical conditions in chronic pericarditis, more especially when it follows an acute affection, are quite different in different cases. It suffices to arrange these differences into two classes, viz., *First.* Absence of liquid effusion, and the pericardial surfaces agglutinated by interposed layers of lymph, of variable thickness, without, or with only imperfectly organized attachment; *Second.* More or less distension of the pericardial sac with turbid serum, puruloid, and even truly purulent liquid. Upon these two classes of anatomical conditions, may be based a division into chronic pericarditis with, and chronic pericarditis without liquid effusion.

In chronic pericarditis without effusion, adhesion of the pericardial surfaces by organized attachment, is prevented by the abundance of lymph. The latter often forms a series of layers which may be successively peeled off from the surface of the heart. These layers are dense, resembling membranous structure, but they are not organized. The pericardial surface beneath presents the appearances of inflammation, and frequently in detaching the layers of lymph, small, circumscribed collections of sero-purulent liquid are discovered. The adherence of the layers of lymph to the visceral and parietal surfaces of the pericardium, and to each other, may be quite firm, but it is only by mechanical agglutination. The deeper layers of lymph are often colored with hæmatin.

A low grade of inflammation is probably kept up by the presence of the lymph in these cases, which acts like a foreign substance, or, successive attacks of subacute inflammation are frequently renewed. More or less enlargement of the heart is apt to follow, and, in some instances, perhaps, atrophy of the organ. These cases are hopeless as regards ultimate, complete recovery, applying the term recovery to the cessation of inflammation, with the occurrence of organized adhesion of the pericardial surfaces, to a greater or less extent.

In chronic pericarditis with effusion, the accumulation of liquid is often much greater than in cases of the acute form of the disease. The pericardial sac, from long-continued distension, yields to the pressure, becomes more or less dilated, and the amount of effusion in some instances is enormous. Perhaps the most remarkable case of large accumulation on record was observed by Prof. Alonzo Clark, and reported by Dr. Swett.[1] Death occurred in ten weeks from the date of the attack, and on examination *post-mortem* "the pericardium was found to occupy the whole anterior part of the chest, pushing the diaphragm downwards so as to form a very large convexity towards the cavity of the abdomen. The liver was pushed downwards so that its upper convex margin reached the margin of the ribs, and both lungs were pushed into the posterior and lateral portions of the thorax. Had the entire contents of the pericardial sac been fluid, it could not have been less than ten pints; but, as it was, there was at least a gallon of clear, yellow serum." Cases in which the accumulation amounts to from two to three pints are not very uncommon. The sac, when dilated much beyond its normal capacity, becomes enlarged disproportionately in width. Its pyriform shape is not preserved, as it is in acute pericarditis. In proportion to its abnormal size, it occupies, of course, space at the expense of the pulmonary organs, and interferes with the thoracic and diaphragmatic movements in respiration.

Chronic pericarditis without liquid effusion is often unattended by symptoms which point to the seat of disease. Acute pain is rarely present. A sense of uneasiness, constriction, or indefinite distress, may be referred to the præcordia; palpitation may be complained of with dyspnœa, on exertion; but there may be entire absence of all subjective symptoms referable to the heart. Feebleness or disordered action sufficient to give rise to symptomatic phenomena denoting plainly cardiac disease may not occur until structural lesions have been induced.

The presence of an abundant liquid effusion will be likely to give rise to symptoms directing attention to the chest, but not distinctive of the disease. The symptoms are essentially those incident to the period of effusion in acute pericarditis, viz., palpitation, feebleness and irregularity of the pulse, præcordial distress, dyspnœa, and tendency to syncope, especially on exertion, lividity, œdema, etc. These symptoms may be less marked than in acute

[1] Lectures on Diseases of the Chest, 1852, p. 394.

pericarditis, although a much larger quantity of liquid may be contained in the pericardial sac, the accumulation taking place more slowly, or greater tolerance being acquired. But even when considerable effusion exists, the disease may be latent as regards subjective and objective symptoms (exclusive of physical signs) distinctly referable to the heart.

The physical signs, when liquid effusion is not present, are not distinctive of existing chronic inflammation. Certain signs may be present denoting union of the pericardial surfaces, but not indicating the mode of this union. These signs will be noticed under the head of pericardial adhesions. Creaking friction-sound is occasionally discovered. The disease, in fact, under these circumstances, frequently does not offer strongly-marked diagnostic phenomena; and, without an acquaintance with the previous history, it is by no means easy to arrive at a positive diagnosis. Knowledge of the fact that acute pericarditis has recently existed, taken in connection with the symptoms and signs, will be a guide to the diagnostician. These cases, when not preceded by acute pericarditis, or when they come under observation after the inflammation has ceased to be acute, are generally overlooked. If, as is often the case, valvular lesions and enlargement of the heart coexist, the symptoms and signs may be referred exclusively to these, pericarditis not being suspected.

An abundant liquid effusion gives rise to physical signs which have been already considered in connection with acute pericarditis. The signs do not differ from those in the latter affection, except so far as they are modified by the existence, often, of a much larger accumulation of liquid than occurs in the acute form of the disease. The space occupied by a largely dilated pericardial sac is, of course, proportionately greater than when the sac is merely distended; and the form of the sac being altered by its greater relative width, the area of dulness on percussion corresponding to the space which it occupies, does not present the pyriform shape characteristic of the outline of dulness in acute pericarditits. The dulness on percussion, in proportion as the amount of liquid is greater, is more marked in degree, approaching more nearly to absolute flatness. The auscultatory signs show removal of the anterior margins of the lung on each side to a greater distance from the median line, the extent of the separation being in proportion to the dilatation of the sac. A friction-sound, in some instances, is discovered, notwithstanding a very large accumulation of liquid. The apex-beat is suppressed;

but a diffused shock may be felt over the præcordia. The latter was observed in the case of enormous accumulation reported by Dr. Swett. It was observed in that case, that the limits of dulness on percussion moved nearly an inch to the right or left, according as the position of the patient was on the right or left side, the extent of the dulness undergoing no change. This variation in situation of the area of dulness, with change of position, in cases of pericardial effusion, has been observed in other cases. Enlargement or bulging of the præcordial region is more apt to be marked in chronic than acute pericarditis, in consequence of the larger collection of liquid. The depression of the diaphragm may be sufficient to cause marked swelling at the epigastrium, and even an unusual prominence of the abdomen. Undulation in the intercostal spaces is oftener observed in chronic than acute pericarditis. In the case reported by Dr. Swett, it was perceived in the epigastrium. The left lung is sometimes pressed upward, to a considerable height, above the clavicle. Dr. Stokes cites a case which came under his observation, in which a tumor was produced above the clavicle sufficiently large to produce great deformity of the neck. This tumor was present for several days; it was increased by coughing, and gave the pulmonary sound on percussion, with vesicular murmur and wheezing rale on auscultation. A similar instance was observed by Dr. Graves. The enlargement of the chest and depression of the diaphragm, in cases of very large effusion, will occasion an obvious restraint of the costal and abdominal movements of respiration.

The differential diagnosis of pericardial effusion, based on the physical signs, involves the same points in chronic, as in acute pericarditis, and these need not be repeated.

The objects of treatment in chronic pericarditis are, the removal, by absorption, of the morbid products, serum and lymph, and the final disappearance of the inflammation. Therapeutical measures having reference to these objects, are, mercurialization, except when contra-indicated by circumstances which have been mentioned; vesication and other modes of counter-irritation, and the use of certain remedies which are supposed to act as sorbefacients, of which the most prominent is iodine. Iodine has been supposed to act efficiently, in some instances, when applied externally. This method is recommended by Dr. Stokes.

But the treatment must be governed, in a great measure, by circumstances which have reference indirectly to the objects just

stated, viz.,the morbid anatomical conditions as respects the presence or absence of liquid effusion; the vital condition of the heart, or, in other words, the weakness of the organ; coexisting affections, and the constitution of the patient. The solid products of inflammation, consisting of thick layers of condensed lymph, cannot be removed by sorbefacient remedies. More is to be expected from efforts to promote the resorption of liquid effusion; but if the pericardial sac be much dilated and the liquid puruloid, the prospect of success is small. In proportion to the weakness of the heart, stimulants are called for. Coexisting affections will claim appropriate attention, and the general condition of the system will be likely to contra-indicate therapeutical measures which tend to impair the vital forces, and, on the other hand, to indicate a sustaining course of treatment.

In cases of large dilatation of the pericardial sac with liquid, which does not diminish under appropriate remedies, and gives rise to distress and danger from compression of the heart and outward pressure on the adjacent parts, the propriety of puncturing the pericardium is to be considered. This operation has been repeatedly performed, with immediate relief of distressing symptoms, apparent prolongation of life, and in some instances perhaps it has been followed by recovery.[1] Assuming the diagnosis to be clear, and that other measures have proved ineffectual, paracentesis is certainly warranted, even as a means of temporary relief. Comparative comfort and postponement of a fatal result may reasonably be expected from the operation. That recovery is not to be looked for in the great majority of cases arises from the almost hopelessness of chronic pericarditis, irrespective of the danger incident to the quantity of liquid accumulation. Further observations may show that the operation is to be resorted to in cases in which the sac is largely distended or dilated, when the distress is not extreme and the danger not imminent. It may perhaps be shown by experience to be applicable to the treatment of acute as well as chronic pericarditis. The success with which paracentesis has of late been employed in cases of pleurisy, by Dr. Bowditch and others, war-

[1] This statement is made with the qualifying word 'perhaps;' for although it is stated that the operation has in some hands been successful, I am unable to refer to reports of cases which afford evidence that recovery was complete. For a *résumé* of cases which have been reported, numbering thirteen, the reader is referred to Bellingham's work, pt. ii. p. 330.

rants a conjecture that the same measure may be extended equally to pericarditis with effusion.

In performing paracentesis of the pericardium, the method practised by Dr. Bowditch in cases of pleurisy is to be preferred. This method consists in the introduction of a small exploring trocar, to the canula of which is attached a suction-pump. The wound made by this instrument is trivial, the liquid may be withdrawn slowly, the quantity regulated by the immediate effects, and the operation repeated as often as may be deemed advisable. The trocar is to be introduced in the fourth or fifth intercostal space between the nipple and the sternum, the patient lying upon the back; the physical signs showing the pericardium to be in contact with the thoracic walls at the point of puncture, and the heart removed from the walls at that situation. M. Aran, of Paris, has reported a case in which a solution of iodine was injected, after the removal of the liquid, with apparent benefit.

PNEUMO-PERICARDIUM AND PNEUMO-PERICARDITIS.

Air or gas gains access within the pericardium by means of fistulous communications with the stomach, œsophagus, or the pulmonary organs; or through wounds of the chest perforating the pericardial sac; and in rare instances it is generated by the decomposition of liquid products within the sac. Inflammation, with more or less liquid effusion, is almost invariably present. The affection is then properly designated pneumo-pericarditis. It is analogous to that variety of pleuritis which is commonly known by the incorrect title—pneumo-hydrothorax. But inflammation is not necessarily present. In a case related to me by Dr. Knapp, of Louisville, to which reference has before been made, a patient was stabbed with a knife, which penetrated the pleural cavity and perforated slightly the pericardium. A splashing sound with the heart's action was immediately heard, which continued for a few days and disappeared. The symptoms and signs, subsequently, did not denote pericarditis, but the patient had pleurisy, which was followed by considerable contraction of the left side. The splashing sound, in this case, was fairly attributable to the presence of air and probably a little blood

within the pericardium. The recovery was complete, and the patient was examined by me some two or three years after the injury. In such a case, the affection, assuming that inflammation was not present, is properly called pneumo-pericardium.

Dr. Stokes relates a case in which the coexistence of liquid and gas was predicated on the peculiar auscultatory phenomena. The account is best given in his own words. "The patient was a young man of lymphatic temperament, who had labored under an attack of acute pericarditis for a few days before I saw him. On my first examination he presented the usual signs of dry pericarditis, with a considerable effusion of lymph of the ordinary consistence. The rubbing sounds, though loud and distinct, had nothing unusual in their character, and the patient suffered but little distress. After two or three days I saw him again, and found that his state had become very much altered. His appearance was haggard and worn, and he complained of extreme exhaustion, which he attributed to a total deprivation of sleep. This was induced by the extraordinary loudness and singular character of the sounds proceeding from the cardiac region; for though up to this period the rubbing sounds were distinctly perceptible by means of the stethoscope, the patient was quite unconscious of their existence. They had suddenly, however, become so loud and singular, that the patient and his wife, who occupied the same apartment, were unable to obtain a moment's repose. On examination, a series of sounds was observable which I had never before met with. It is difficult or impossible to convey in words any idea of the extraordinary phenomena then presented. They were not the rasping sounds of indurated lymph, nor the leather creak of Collin, nor those proceeding from pericarditic with valvular murmur, but a mixture of the various attrition murmurs with a large crepitating and a gurgling sound, while to all these phenomena was added a distinct metallic character. In the whole of my experience I never met with so extraordinary a combination of sounds. The stomach was not distended with air, and the lung and pleura were unaffected, but the region of the heart gave a tympanitic *bruit de pot fêlé* on percussion; and I could form no conclusion but that the pericardium contained air in addition to an effusion of serum and coagulable lymph. In the course of about three days the signs of air disappeared, leaving the phenomena as they were at the first period of the case. The convalescence of this patient was slow, and the rubbing sounds continued

for an unusual length of time. His recovery was ultimately perfect."[1]

Dr. Stokes, in connection with this case, cites two additional cases, one reported by Dr. Graves, and the other communicated by Dr. B. McDowel. In Dr. Graves' case, pericarditis was induced by the opening of a hepatic abscess into the pericardial sac. The case proved fatal, and after death it was ascertained that the abscess also communicated, through a fistulous opening, with the stomach. The gas contained within the pericardium was derived from the stomach passing through the hepatic abscess. The patient presented over the præcordia friction-sounds, with an occasional metallic click, giving the idea of a fluid dropping in the pericardium. Afterwards the sounds assumed the character of an emphysematous crackling. In Dr. McDowel's case, a fistula was found, after death, to have become established between the pericardial sac and a small anfractuous cavity in the right lung. A current of air through the trachea was observed to rise through the fluid contained in the pericardial sac, and the latter, when opened, contained air. Over the left side of the chest, in this case, auscultation discovered metallic tinkling, and splashing of fluid caused by the action of the heart.

In a case reported by Dr. Walshe, in which a communication existed between the œsophagus and pericardium, produced by the effort to swallow a knife, tympanitic resonance on percussion over the præcordia was marked, but neither a splashing noise nor metallic tinkling were observed. A distinctive phenomenon in this case "consisted in the change of position of tympanitic and dull percussion-sound, within the area of the cardiac region, according as the posture of the patient was changed from one to the other side."

These cases are of much interest as showing the physical signs distinctive of the presence of air or gas and liquid within the pericardium. The auscultatory signs which may be expected to be present are, metallic tinkling sounds, and a splashing or gurgling noise, produced by the action of the heart. Their connection with the heart is to be determined, if there be room for doubt, by requesting a momentary holding of the breath. They are not, however, invariably present, as shown by the case reported by Dr. Walshe. Tympanitic resonance on percussion over more or less of the præcordia is marked. In the case observed by Dr. Stokes, a distinct *bruit de pot fêlé* was observed; and in Dr. Walshe's case

[1] On Diseases of the Heart and Aorta, Am. ed., p. 38.

variation in the relative position of tympanitic resonance and dulness, with change of posture. The production of a peculiar noise, so loud as not only to be heard by the patient and others, but to prevent persons from sleeping in the same apartment, is a remarkable and highly distinctive feature in Dr. Stokes' case.

The physical signs, in connection with the history and symptoms, seem to be amply sufficient for the diagnosis. There is a possibility that considerable distension of the stomach with gas and liquid, may give rise to acoustic phenomena resembling those produced in some cases of pneumo-pericardium. But the evidences of pericarditis with effusion, under these circumstances, will be wanting. Cardiac gastric sounds, probably, require for their production that the pericardial sac shall be free from liquid. Again, metallic tinkling, and, possibly, splashing sounds may be produced by the action of the heart in some cases of pneumo-hydrothorax; but it is sufficiently easy to exclude the latter affection by the absence of its diagnostic signs.

The treatment of this variety of pericarditis does not claim distinct consideration.

PERICARDIAL ADHESIONS.

Inflammation of the pericardium, ending in recovery, involves, as has been seen, the formation of new tissue which often serves as a medium of permanent union of the opposed pericardial surfaces. Pericarditis, when general, *i. e.*, when the inflammation extends over the whole, or the greater part of the membrane, is followed by this result, as inflammation of the pleura is followed by pleuritic adhesions. The pericardial adhesions now referred to, differ from those which have been considered as incident to a variety of chronic pericarditis. The latter are due to a stratum of lymph interposed between the surfaces of the pericardium, to which each pericardial surface becomes agglutinated. The lymph is unorganized, and is, in fact, equivalent to a foreign substance, at once separating and binding together, mechanically, the parietal and visceral portions of the pericardium. Under these circumstances, the pericardium is rarely, if ever, in a healthy condition. In adhesions by means of new tissue, the mode of union is quite different. It is by an or-

ganized attachment. The new structure, when formed, becomes thereafter an integral portion of the organism. These adhesions are not incompatible with a healthy state of the pericardial membrane; they do not necessarily constitute a disease, although they are the effects of disease. They become more and more firm with age. Some idea may be formed of the length of time since their formation, by the force required for their separation. It is customary to speak of them as more or less ancient. It is doubtful whether recovery from general pericarditis ever takes place without leaving more or less of these effects. The adhesion may be general or partial; in other words, the surfaces may be united over the whole heart, or only over a portion of the organ. General adhesion appears to be of much more frequent occurrence than partial. Of 70 cases analyzed by Louis, the adhesions were general in 60, and partial in 10; and of 86 cases analyzed by Dr. Chambers, 51 were universal, 4 nearly so, and 29 partial.[1] When the adhesion is general and close, the pericardium seems to be wanting, and it is conjectured that some of the cases reported by the early anatomists of absence of the pericardium, may have been cases of this description.

Pericardial adhesions are found, on examination after death, associated, in a certain proportion of cases, with valvular and other cardiac lesions. They are also found, not infrequently, when there had been no suspicion of cardiac disease. They denote, of course, that pericarditis has existed at some period during life, and this period may have been more or less remote from the time of death. They constitute, in a certain proportion of cases, the only abnormal condition which the heart presents. The practical questions connected with the subject are: What effects do they produce upon the heart and circulation, and how is their existence to be ascertained during life? These questions, it is obvious, are of considerable importance practically. They suggest the most convenient arrangement for the consideration of the subject.

What effects are produced by pericardial adhesions upon the heart and circulation?

Pathologists, for the most part, up to the present time, have regarded adhesions of the pericardium as constituting a very serious

[1] Decennium Pathologicum, Brit. and For. Med.-Chir. Rev., vol. xii. 1853. Also Bellingham, op. cit.

abnormal condition. Laennec held a contrary opinion, considering them as often harmless. Among recent writers on diseases of the heart, Bouillaud concurs in the opinion of Laennec; but the doctrine inculcated by Hope is, that they inevitably lead to enlargement of the heart, and, sooner or later, to a fatal result. Investigations since the publication of the last edition of the treatise by Hope show, conclusively, that this distinguished author was led to exaggerate the evils and dangers attendant on the remote consequences of pericarditis.

Pericardial adhesions, general, and evidently of long standing, are found, not infrequently, when, in all other respects, the heart presents a normal, healthy appearance. Cases exemplifying the correctness of this statement have been reported by Bouillaud, Stokes, King, Barlow, W. T. Gairdner, and others.[1] The following instance, which I find among my recorded cases, will serve as an illustration: A male patient, aged 35, was admitted into the hospital, in a state of unconsciousness, and died fifteen hours after admission. The previous history was not ascertained. On examination after death, morbid appearances were found denoting meningitis. The heart presented universal, old adhesions. Considerable force was required in separating the pericardial surfaces. The organ was apparently not enlarged. It weighed 10½ ounces. The walls and cavities were normal, and the muscular substance was not altered in color or consistence. In this instance, that the adhesions were of ancient date is inferred from their firmness. Dr. Stokes refers to a case which came under his observation, in which, death occurring seven years after an attack of pericarditis, the patient, for several years, exhibiting no symptoms of heart disease, the pericardial sac was found to be obliterated, and the heart otherwise in a perfectly natural condition. The occurrence, in even a small number of cases, of adhesions, which, after several years of exemp-

[1] For the convenience of those who may wish to consult the authors named, the references are subjoined as follows: Stokes on "Diseases of the Heart and Aorta." W. King on "the Harmlessness of Adherent Pericardium," *London Lancet*, Nov., 1845. Article by Dr. Barlow in "Gulstonian Lectures." Dr. W. T. Gairdner on "The Favorable Terminations of Pericarditis, and especially in Adhesion of the Pericardium, with cases illustrating its Secondary Effects on the Heart," *Edinburgh Monthly Journal of Medical Science*, 1851. Bouillaud, in *Leçons Cliniques sur les Maladies du Cœur*, etc., Paris, 1853. The author last named states that he has met with more than fifty examples of pericardial adhesions in persons who had enjoyed good health for a long period, and who died with various affections foreign to the heart.

tion from all cardiac trouble, are found not to be accompanied by any other abnormal condition of the heart, suffices to show that they do not necessarily give rise, as stated by Hope, to serious effects upon the heart and circulation.

The foregoing conclusion, however, may be correct, and yet pericardial adhesions exert more or less agency in the development of cardiac disease in a certain number of cases. It is, therefore, an important object to determine the proportion of cases in which these adhesions are found to exist independently of other abnormal conditions of the heart. With reference to this point, Dr. Gairdner has analyzed 15 cases in which adhesions were found after death, the patients dying from various diseases. Of these cases, in 10 the heart was not enlarged, nor otherwise diseased. Of the remaining 5 cases, the heart was enlarged in all; but in 2 of the latter cases, valvular lesions coexisted; and in 2 the adhesions were not general, but partial. These 15 cases were collected by Dr. Gairdner from the records of 500 miscellaneous post-mortem examinations performed in the Edinburgh Infirmary, the cases of adherent pericardium only being selected in which the adhesions were so considerable and so situated as to restrain the movements of the heart. An analysis of 90 cases of adherent pericardium, collected from museums and catalogues by Mr. Henry Kennedy, of Dublin, yields results somewhat different from those obtained by Dr. Gairdner. Of these 90 cases, the heart remained healthy till death, in 34, and was enlarged in 51.[1] From this collection, all cases of valvular lesions were excluded. Of four cases of general adhesions disconnected from valvular disease, of which I have notes (and also the hearts, in my cabinet), in one only was the heart normal in other respects. In three cases there existed a moderate degree of enlargement.

In view of the discrepancy of the results of different analyses, the precise ratio of instances in which pericardial adhesions are found not to be accompanied by other abnormal conditions, which may be considered as consecutive, is not, as yet, determinable, but we are warranted in concluding that the proportion is at least one-third, excluding cases in which they are associated with valvular lesions.

It is evident that, in order to determine more fully the effects of adhesions on the heart, it is desirable to know, in the cases in

[1] Edinburgh Med. Journal, 1858.

which they are found not to be associated with any other abnormal condition, how long they have existed prior to death. Often this cannot be definitively ascertained. A collection of cases in which the length of time that had elapsed after the occurrence of pericarditis, is known, and the heart examined after death, would be highly valuable with reference to the question under consideration.

Adhesions existing in connection with valvular lesions, are properly excluded from the cases analyzed with a view to determine the effects of the former upon the heart. Valvular lesions, as is well known, in the great majority of cases, lead to cardiac enlargement, so that when these lesions coexist with adhesions, and the heart is found to be enlarged, it is fair to attribute the enlargement to the affection of the valves. And when valvular lesions and adhesions are combined, it may be concluded that the inflammatory affections giving rise to both, occurred at the same time, inasmuch as clinical observation shows pericarditis in the majority of cases to be conjoined with endocarditis. It is also to be considered that the association of adhesions with enlargement of the heart (valvular lesions not being present) does not prove that the latter is an effect of the former. Other causes may have given rise to the enlargement, and the association may be merely a coincidence in some cases. Again, the enlargement may have proceeded not from the adhesions, but as a remote effect of the pericardial inflammation on the substance of the heart. The adhesions and the enlargement, in these cases, are coinciding effects of a common cause, viz., inflammation of the pericardium without any causative dependence on each other. Still farther, it is to be borne in mind that a moderate enlargement of the heart is not necessarily a serious affection. And, in point of fact, pericardial adhesions associated with a certain amount of enlargement, without the coexistence of valvular lesions, are often, if not generally, found after death in cases in which the symptoms had not denoted any cardiac affection, and death was owing to diseases having no apparent reference to the condition of the heart. In view of the several considerations just presented, it seems to be a logical conclusion that pericardial adhesions do not involve serious consequences to the extent which might be inferred from the statistics of Dr. Kennedy, showing that adhesions and enlargement are found after death to be associated in two-thirds of the cases examined, exclusive of the cases of valvular lesions. To these considerations it may be added that

adhesions, enlargement, and valvular lesions combined, are sometimes borne for a long period. This fact is illustrated by the following case: A male patient, aged 68, was admitted into the hospital with advanced cardiac disease, and died the day after his admission. It was ascertained that 38 years had elapsed since an attack of acute rheumatism. The pericardium was found, on examination after death, to be universally and closely adherent by firm and evidently very old adhesions. The heart weighed 46 ounces. The enlargement was due mainly to hypertrophy and dilatation of the left ventricle and auricle, but the whole organ was increased in size. The curtains of the mitral valve were thickened, contracted, and the valve evidently insufficient. The aortic valve was sound.

The doctrine inculcated by Hope is, that pericardial adhesions invariably lead to enlargement of the heart. It has been seen that clinical facts abundantly disprove this doctrine. On the other hand, it has been recently maintained that the effect of adhesions is precisely the reverse of this; that they tend to produce atrophy of the organ. This view was first taken by Dr. Chevers.[1] It is adopted by Dr. Barlow and W. King in the papers already referred to. Professor Smith, of Dublin, thinks that he has found atrophy and hypertrophy to coexist with adhesions in about an equal proportion of cases. Dr. Stokes advocates this view on the ground of analogy with the apparent effects of pleuritic adhesions on the lungs, and of mechanical restraint on the voluntary muscles. Dr. Hope accounted for the production of hypertrophy on the principle that the heart, being mechanically restrained, was thereby excited to increased power of action to overcome the restraint; he applied, in other words, the principle on which valvular obstruction leads to hypertrophy. But the analogy does not hold good. As remarked by Dr. Stokes, "In adhesion, the normal condition of the muscle is interfered with and the contraction diminished; while in valvular obstruction, the muscle being free to act, increases in power, just as the voluntary muscles do when trained by exercise." Atrophy is supposed to be produced as an effect of pressure, which not only restrains the movements of the heart, somewhat like a bandage applied over the muscles of an extremity, but by interfering with the free supply of blood to the substance of the organ. This doctrine, however rational, with our present knowledge must

[1] Guy's Hospital Reports, vol. vii.

be regarded as hypothetical; and, as a matter of observation, enlargement is much oftener found associated with pericardial adhesions than an abnormal diminution of the volume of the heart. Dr. Kennedy found evidence of atrophy in only five of the ninety cases analyzed by him. True atrophy, *i. e.*, in which the organ is reduced in volume, as is well known, is one of the rarest of cardiac lesions.

In conclusion, while it is not to be denied that pericardial adhesions do contribute to enlargement of the heart in a certain proportion of cases, nor that they may also, in rare instances, tend to an opposite result, they exist not infrequently for a long period without being followed by any appreciable morbid condition, and their tendency to the production of either organic change or functional disturbance is vastly less than was supposed by Hope. With this general view of the effects upon the heart and circulation, the remote consequences of pericarditis, so far as the pericardium is concerned, need not occasion much apprehension. It is not improbable that obliteration of the pericardial sac after acute pericarditis is, in some sense, a conservative result, preventing, in some instances, persistence of the inflammation in a chronic form. And it is highly probable that when pericarditis is followed by cardiac enlargement, exclusive of the cases in which valvular lesions coexist, the enlargement is due more to the condition of the muscular substance of the heart, produced directly by the inflammation, than to the effect of the adhesions. Assuming this to be the view most consistent with our present knowledge, its practical importance, as regards its bearing on the prognosis and management after recovery from pericarditis, is sufficiently obvious.

How is the existence of pericardial adhesions to be ascertained during life?

If it be true that adhesions may remain innocuous for an indefinite period, and that they rarely, if ever, of themselves give rise to serious consequences, the importance of ascertaining their existence during life is less than it would be were their effects on the heart and circulation more important. Assuming the correctness of the general view of these effects which has been presented, I shall not dwell long on the present division of the subject.

Symptoms referable to the heart and circulation are not distinctive of adhesions. Whatever evidence is available must be derived

from physical signs. The diagnosis cannot always be made with positiveness; but, in a certain proportion of cases, the signs may be relied upon with much confidence, especially if the previous history render it highly probable that pericarditis has existed at a period more or less remote. The diagnostic points furnished by exploration require that the adhesions shall be nearly or quite universal, and they are more marked when, in addition to union of the pericardial surfaces, there exists firm adhesion of the exterior of the pericardium to the parietes of the chest.

Percussion shows that the heart is in contact with the chest over a larger space than normal; in other words, the area of the superficial cardiac region is enlarged. But as this occurs whenever the heart is increased in volume, alone, it is not distinctive. Another sign obtained by percussion is significant. It is, persistence of the dulness within the same area in different positions of the body, and when the patient takes a deep inspiration. This shows that the heart is fixed, and that external adhesions prevent the lung from overlapping the organ, even when expanded by a forced effort.

Auscultation concurs with percussion in showing that the lungs do not extend over the heart on a full inspiration, provided the pericardium be united to the chest by pleuritic adhesions. This method of exploration furnishes no other diagnostic points. Clinical observation has not established any peculiar modifications of the heart-sounds. On theoretical ground, it is probable that the element of impulsion in the first sound is weakened, but the variations in this respect in health and disease are such as to render this alteration of slight import.

Palpation furnishes important signs. The apex-beat is frequently suppressed. It is not true that it is invariably wanting, even when universal and close adhesions exist. And, on the other hand, the apex-beat is not only suppressed in connection with different forms of disease, but it is not felt in all healthy persons. Alone, absence of the beat is by no means distinctive, but it is significant when taken in connection with other signs. Its suppression is accounted for by the fact that pericardial adhesions interfere with the elongation and locomotion of the heart's apex, more particularly the latter. When not suppressed, the beat may be felt higher than its normal position, viz., in the fourth instead of the fifth interspace, while the body is in a vertical position. This change of position, in connection with other signs, has considerable significance. If, however,

the heart be much enlarged, the apex may be lowered, notwithstanding the adhesions.

Impulses may be felt in the intercostal spaces above the point of apex-beat, but this is not infrequently observed in cases of simple enlargement. Successive movements in different intercostal spaces, presenting an appearance of undulation, can hardly be considered as a sign of adhesions, if enlargement be present.

The apex-beat undergoes but little alteration in its position with change of posture. This sign has considerable significance. In health, the position of the beat is removed to the left, from half an inch to an inch, by changing the posture from that on the back to that on the left side. Pericardial and pleuritic adhesions prevent this lateral movement of the heart's apex. I have observed, in a patient supposed to have adhesions, that the impulse not only remained in the same position, but retained the same force, the patient lying on the right side, as when lying on the back. In health, the impulse is either moved to the right by this change of posture, or, more frequently, lost. Another point pertaining to the apex-beat is its preserving the same position with the different acts of respiration. It is not depressed by a forcible inspiration, nor raised by a forcible expiration, to the same extent as in health.

A jogging or tumbling motion of the heart, as perceived by the hand, was considered by Hope as a distinctive sign. But violent and disturbed rhythmical action is not only observed in cases of enlargement and of merely functional disorder, but tranquil regularity of the heart's movements is perfectly compatible with universal and close adhesions. Nor is a sensation, communicated to the hand, as if the heart were restrained, or were struggling against an obstacle, of which Bouillaud speaks, to be relied upon.[1] Such a sensation must involve a preconceived idea that adhesions exist.

Inspection furnishes signs which are the most distinctive, viz., retraction of the intercostal spaces, and depression of the epigastrium to the left of the xiphoid cartilage, occurring synchronously with the ventricular systole. The depression movement of the epigastrium is due to the attachment of the base of the pericardium to the cordiform tendon of the diaphragm; the retraction movement of the intercostal spaces is caused by the drawing in of these spaces when the ventricles contract, and is most marked when the peri-

[1] "On sent à la main que le jeu du cœur est embarrassé, difficile, etc." Leçons Cliniques, 1853.

cardium is attached to the parietes of the chest. One or two, and possibly three, of the intercostal spaces may present retraction, and in some instances the ribs are also retracted. Depression of the epigastrium may be present alone, or associated with intercostal retraction. In some instances the xiphoid cartilage, and even the lower portion of the sternum, is drawn inward, apparently with considerable force. These signs, when present in a marked degree, and especially in combination, are highly distinctive.

To recapitulate the physical signs denoting pericardial adhesions, they are as follows: the area of præcordial dulness on percussion remaining unaltered in different positions of the body, and not affected by a deep inspiration; the limits of the respiratory murmur not affected by a deep inspiration; the apex-beat often suppressed, and, if not suppressed, often raised above its normal position; the apex-beat, if felt, unaffected, or affected but slightly, by changes of posture from the back to either side, and by forcible inspiration or expiration; retraction of one or more intercostal spaces, together with the ribs in some cases, and depression of the epigastrium synchronously with the ventricular systole.

These signs strengthen each other by combination. The more are combined, the greater the significance of each. Not one, however, is constant, and all may be wanting in cases of pericardial adhesions. They are rarely, if ever, marked, unless the pericardium be attached to the thoracic walls by pleuritic adhesions, and these do not always coexist with union of the pericardial surfaces. A positive diagnosis, therefore, is only practicable in a certain proportion of cases. If it be known that a patient has had, at some past period, an attack of pericarditis, this fact renders less physical evidence necessary for the diagnosis than if the previous history contained no information with respect to that point. The fact of the patient having had acute rheumatism adds weight to the conclusion drawn from the physical signs. Contraction of the chest, limited to the præcordia, is also a collateral point of evidence, as showing that pericarditis has existed.

CHAPTER VIII.

INFLAMMATORY AFFECTIONS OF THE HEART. ENDOCARDITIS. MYOCARDITIS.

Endocarditis—Definition—Anatomical characters—Pathological relations and causation—Symptoms—Physical signs—Diagnosis—Prognosis—Treatment—Myocarditis.

ENDOCARDITIS.

INFLAMMATION of the endocardium, the membrane which lines the cavities of the heart and is duplicated to cover the valves, is called endocarditis. This name originated with Bouillaud, who was the first to recognize clearly the occurrence of inflammation in this situation. Recent clinical researches have shown that this disease, the nosological existence of which dates from a little more than a quarter of a century ago, is by no means infrequent. It occurs as a complication of acute rheumatism, in a large proportion of cases. The knowledge of its frequent coexistence with this affection, is one of the most important of the developments of modern medicine. The remote effects of endocarditis, as involved in the valvular lesions which have been considered in a former chapter, invest the disease with much importance. Inflammation here, as in other situations, may be acute, subacute, and chronic; but it is hardly practicable to make these distinctions, clinically, and hence it suffices to consider the subject under the head, simply, of endocarditis. In the consideration of this subject, the same divisions will be adopted as in treating of acute pericarditis, viz., the anatomical characters of the disease, its pathological relations and causation, its symptomatic phenomena, its physical signs, the diagnosis, the prognosis, and the treatment. These divisions will be taken up in the order in which they have just been named.

Anatomical Characters of Endocarditis.

Endocarditis is seated, in the vast majority of cases, in the cavities of the left side of the heart. The lining membrane of the right auricle and ventricle is rarely inflamed. When inflammation does exist in the cavities of the right side, it is also present, almost invariably, in those of the left side. The instances of endocarditis limited to the right cavities, are exceedingly rare. All portions of the endocardial membrane are not equally subject to inflammation. The portions covering the valves, and lining the orifices are especially prone to become inflamed. Endocarditis is generally limited to these situations. The membrane here is most exposed to the action of the blood-currents; the valvular portion is in constant motion, and considerable tension, or stretching, must take place with each ventricular systole. But another, and perhaps a stronger, reason for the limitation of inflammation to these situations, is derived from the fact that the membrane is here underlaid by fibrous tissue, while in other portions it is in close proximity to the muscular walls of the heart. The rule as regards the greater liability of the left side of the heart, to endocardial inflammation, is applicable after birth, but probably does not hold good during intra-uterine life. There are grounds for the belief that the fœtus *in utero* is subject to endocarditis, and that at this period the inflammation is generally seated in the right side of the heart. The malformations which have been considered in a previous chapter, are in a measure thus accounted for.

Opportunities for inspecting the morbid appearances during the progress of endocarditis, are not often presented. The disease very rarely proves fatal. It is not the immediate danger, but the remote consequences which render it a formidable affection. The anatomical characters which have been observed in the occasional instances in which death has occurred when inflammation existed, embrace here, as in other situations, redness from vascular injection, alterations in the membrane itself, and the presence of inflammatory products.

Redness due to endocarditis is caused by injection of the vessels which ramify in the areolar tissue beneath the membrane. It is not always found when inflammation undoubtedly existed at the time of death. It may disappear as a post-mortem change. On the other hand, mere redness is by no means adequate evidence of the

existence of inflammation. It is often observed in the cavities of the heart, and in the large vessels, as an effect of the imbibition of hæmatin dissolved out of the red globules of the blood which these cavities and vessels contain after death. Under these circumstances, it is a cadaveric staining of the membrane, and is an effect of the decomposition of the blood. It is observed frequently in post-mortem examinations, more especially when these are made two or three days after death, or when the warmth of the weather favors putrefactive changes; and it is a post-mortem condition found after certain diseases in which the blood undergoes notable changes prior to death. The redness from imbibition is distinguished from that due to inflammation, by the following points of difference: It is not an arborescent, but an uniform redness, and when examined with a lens, injected vessels are not visible. It is deeper or of a darker color than inflammatory redness. It is more likely to be observed in the right than in the left side of the heart, a larger quantity of blood usually remaining in the right cavities after death. It is not limited to the valves or orifices, and usually extends into the arteries where it is more conspicuous than in the cardiac cavities. The redness is most marked in the dependent parts of the heart and vessels. It is readily removed by maceration, which is not true of inflammatory redness. Exclusive of the discoloration, the membrane preserves its natural appearance; it is firm and polished as in its normal condition, and does not present any of the inflammatory products. Whatever may be the characters pertaining to redness, if the membrane be normal in all other respects, and the products of inflammation wanting, the evidence of the inflammatory state is insufficient.

Anatomical changes in the membrane are much more distinctive of inflammation than redness. These changes are, loss of the transparent, smooth, polished appearance which this structure presents in a healthy state, instead of which it becomes opaque, rough, velvety, and felt-like; more or less swelling and softening; brittleness of the subjacent areolar tissue, in consequence of which it is more easily detached than in its normal condition. Anatomical changes are often found which are due to ancient inflammation, and to morbid processes not inflammatory, such as atheromatous deposit and hypertrophy of the endocardium. These changes are, of course, to be distinguished from those which denote endocarditis existing at the time of death.

Other characters relate to the products of inflammation. As

regards these, the endocardium, although resembling in structure serous membranes, differs from the latter in not being a shut sac, within which the inflammatory products are collected and retained for a greater or less period. Morbid deposits are liable to be detached, washed away by the currents of blood, and carried along with the circulation. The endocardium differs from serous membranes in another important point, viz., it is in contact with the blood itself; and while this fluid, in motion, detaches and removes deposits, it may also furnish them by yielding a portion of its fibrin which undergoes coagulation.

The products of inflammation in endocarditis may be derived from two sources, viz., the coagulation of fibrin, just alluded to, and the exudation of lymph occurring here precisely as in serous inflammations. In the one case they are derived from the blood within the cavities of the heart, and in the other case, from the blood in the vessels situated in the areolar tissue beneath the endocardium. The extent to which deposits found after death are derived from these two sources, respectively, has given rise to much discussion among pathologists. The question is one not easily settled with exactness. The most rational view, with our present knowledge, is, that both sources are generally involved, and in different proportions in different cases. Exudation of lymph from the blood contained within the vessels, doubtless takes place here, as in analogous structures when inflamed. The deposit from this source occurs on the free surface of the membrane, and beneath the membrane. If not detached and washed away by the blood-currents, the lymph exuded on the free surface remains attached to the membrane. It is not infrequently found adherent in membranous-like layers as in cases of pericarditis or pleuritis, but not in the same abundance. The roughness produced by the exudation of lymph or the alterations of the membrane, attracts, as it were, the fibrin from the blood, and leads to its precipitation in a coagulated state. The deposit is thus augmented from this second source. The lymph is equivalent to a foreign substance, and becomes coated with fibrin, like the threads passed through arteries in Dr. Simon's experiments. An increased proportion of fibrin in the blood, which characterizes in a marked degree acute rheumatism, probably favors the deposit from the latter source. This twofold origin of the products of inflammation is important in its bearing on the treatment of endocarditis.

The inflammation affecting especially the valves and orifices of

the left side of the heart, morbid deposits, as well as other anatomical changes, are usually found in these situations. The deposits consist of lymph upon and beneath the endocardium, rendering the membrane opaque and apparently thickened, and forming vegetations which are composed partly of lymph, and in part of coagulated fibrin. The latter are found more especially either at the base or free extremities of the valves, and they are most apt to occur on the surface of the valves exposed to the direct current of blood, *i. e.*, on the auricular aspect of the mitral, and on the ventricular aspect of the aortic, valve. Deposits consisting of lymph frequently assume the form of small granular masses or beads, varying in size from that of a pin's head to a millet-seed, studding the margins of the curtains of the mitral valve and the tendinous cords, and fringing the crescentic extremity of the fibrous portion of the segments of the valve of the aorta. These deposits are removed with slight force after recent endocarditis, and the surface beneath may be roughened, excoriated, and thickened, or changes in the appearance of the membrane may not be marked. The deposits, however, are often larger, forming excrescences of the size of a pea, and even considerably larger than this. These consist usually of the smaller bead-like granulations of lymph, which have become coated by the accretion of fibrin. The latter constitutes the larger portion of their substance.

The exudation of lymph is unequivocal evidence of the existence of endocarditis. The deposit of fibrin has not this significance in an equal degree. The latter, according to the views of some distinguished pathologists,[1] may occur solely as a result of an excess in the fibrinous constituent of the blood, or of some change in consequence of which the blood does not retain the fibrin in a liquid form, as in health. The presence, therefore, of deposits in the form of vegetations is not positive proof that endocarditis has existed, unless they consist, in part at least, of exuded lymph. The appearances of the deposits do not always suffice for the discrimination between fibrin and lymph, and hence the deposits are not to be considered as anatomical characters which, alone, are absolutely reliable. As criteria of inflammation, they are inferior to the changes which the endocardial membrane presents. But the latter, in general, coexist, to a greater or less extent, with inflammatory deposits. The deposit of fibrin in cases of old valvular lesions, and when the

[1] *E. g.*, Rokitansky, *vide* Path. Anat.; Dr. Fuller, *vide* work on rheumatism.

endocardial membrane has undergone changes incident to atheroma or other processes not inflammatory, does not constitute evidence of recent endocarditis. Roughness of the membrane, irrespective of existing inflammation, suffices to determine this deposit, especially when the condition of the blood is such as to favor the separation and coagulation of fibrin.

Other morbid changes which may occur during the progress of endocarditis are, loss of substance, or destruction of portions of the membrane, by ulceration or erosion; perforation of the valves; lacerations; and, according to Bouillaud, occasionally gangrene. These are rare occurrences as immediate results of the inflammatory processes. Lacerations and erosions are the least infrequent. Adhesion of the valves to each other, to the walls of the heart, or, in the case of the semilunar valves, to the inner surface of the artery, are to be included among the comparatively rare anatomical changes incident to recent endocarditis. Other effects are true morbid growths, as distinguished from the vegetations which consist of either lymph or fibrin, or both combined.

The opportunity of inspecting the morbid appearances during the different stages of the progress of endocarditis, as already stated, is rarely presented, inasmuch as the disease seldom proves fatal. For this reason the appearances found after death in the experiments made by Dr. Richardson, in which endocarditis was artificially induced in inferior animals by the introduction of lactic acid into the blood, are of much interest and value.[1] In sixteen experiments on dogs, cats, and rabbits, endocarditis was invariably produced when a certain quantity of lactic acid, largely diluted, was injected into the peritoneal cavity. If the animals died or were killed at a period when the symptoms denoted commencing inflammation, the endocardial membrane presented a brilliant vermilion color; it had a velvety or villous appearance, and beads of lymph or fibrin were abundant. At a somewhat later, but still early, period the auriculo-ventricular valve became thickened and œdematous. The writer says: "I have seen the segments of the tricuspid valve fixed in this swollen condition, resembling each an injected uvula, and lying so close to each other that, when the heart was contracting, they must have cushioned against each other, thus fulfilling their office of preventing regurgitation passively, *i. e.*, without tension or move-

[1] An Experimental Inquiry on Endocarditis, by the Synthetical Method. By Benjamin W. Richardson, M. D., etc. Contained in *Virginia Medical Journal*, March, 1859. See also appendix to Prize Essay, London, 1858.

ment. In this œdematous stage, if the valve be pricked with a needle, a clear white lymphy fluid exudes, and by frequent pricking the valve structure, emptied of its effusion, collapses and assumes a flaccid condition." At a later period "the valves remained thickened, but the red color and œdematous state were both reduced. Beneath the endocardial surface of the valve there was a paleness as from coagulated effused lymph. If the needle be applied now, there is no exudation; the valve has some limited play, unless it is bound down by adhesion, and its structure is firm. Beads which generally fringe the margin of the valves all around, from being œdematous prominences in the earlier stages, are pearly looking, and are moderately firm." Still later, the writer describes the valves as shrunken, having regained imperfect play, but still thickened and unyielding. It is probable that this account of the morbid appearances in inferior animals may be applied analogically to endocarditis in man, during periods of the disease when the anatomical characters can be studied in only the very rare instances in which a fatal result takes place, and when a sufficient time has not elapsed after death for important post mortem changes to have ensued. These experiments will be again referred to in connection with the causation of endocarditis.

A point observed by Dr. Richardson with regard to the situation of the anatomical changes in artificially induced endocarditis may be here mentioned. These changes were mostly confined to the auriculo-ventricular valves. He states: "A very slight thickening, not sufficient at any time to interfere seriously with their duties, is all I have ever observed in the semilunar valves on either side."

The remote effects of endocarditis are vastly more serious than the immediate anatomical changes. The latter lay the foundation of the various valvular lesions which were considered in Chapter III. These lesions are slowly induced in consequence of the presence of the morbid deposits, their progressive increase by accretion from the precipitation of fibrin, the process of calcification, etc., together with the softening, friability, and sometimes solution of continuity of the endocardium and the subjacent tissue. Insufficiency of the valves and contraction of the orifices are ulterior consequences, giving rise to enlargement of the heart and other pathological effects which have been treated of in preceding chapters. With reference to their eventuation in valvular lesions involving either obstruction or regurgitation, or both, the immediate anatomical changes in endocarditis are of great importance.

In a very large proportion of cases, valvular lesions owe their origin to these changes. Hence, a person attacked with endocarditis is liable, at a period more or less distant, perhaps after many months or years, to fall a victim to organic disease of the heart. The rapidity and extent of the remote effects will be proportionate, other things being equal, to the amount of deposits and other changes which remain after the endocardial inflammation has ceased. In this respect different cases doubtless vary. It is possible for the fibrin and lymph deposited upon the free surface of the membrane to be gradually washed away by the blood-currents, the inflammatory products beneath the endocardium removed by absorption, and the normal condition of the valves restored, so that no ulterior evils follow the disease at any period, however remote. This happy termination, if it ever occurs, is a rare exception to the general rule.

Pathological Relations and Causation of Endocarditis.

Endocarditis resembles pericarditis in the infrequency of its occurrence as an idiopathic affection. It occurs, however, independently of other affections, oftener perhaps than is generally supposed, its latency as regards symptoms being such that it is apt to be overlooked. In the great majority of the cases in which its existence is ascertained, it is associated with acute articular rheumatism. It is comparatively a very rare affection as occurring in other pathological connections. Rheumatic endocarditis is sufficiently common for cases to fall frequently under the observation of the medical practitioner. It is perhaps not far from the truth to say that, in a case of acute articular rheumatism, the chances are about equal that the endocardial membrane will become involved. Endocarditis and pericarditis are not infrequently associated in cases of rheumatism. The combined affections are designated *endo-pericarditis*. Rheumatic pericarditis very rarely exists without endocarditis. But the converse of this statement does not hold good; endocarditis often exists without being associated with pericarditis. The statement by Hope that endocarditis exists without pericarditis much oftener than pericarditis without endocarditis I believe to be correct, although an opposite opinion is held by Dr. Stokes. With reference to the frequency of endocarditis in acute rheumatism, and the relative proportion of cases in which endocar-

ditis, pericarditis, and endo-pericarditis, respectively, occur in the cases of rheumatism with cardiac complication, the following statistics may be cited: Of 474 cases collected from various sources and analyzed by Dr. Fuller, endocarditis existed in 214, the ratio being as 1 to every 2.25 cases. These cases were reported by four different observers, and the analysis of each collection gives not far from the same ratio as when they are analyzed collectively. Of 204 cases of rheumatism with cardiac complication of some kind, endocarditis existed in 138, pericarditis in 19, and endo-pericarditis in 38. The accuracy of these statistics is, of course, based on the practicability of determining the coexistence of endocarditis in cases of rheumatism, and there are certain difficulties in the way of arriving at a positive conclusion with respect to this point in some cases, as will appear in connection with the subject of the diagnosis.

The portion of the endocardial membrane which covers the mitral valve is affected oftener and to a greater extent than the portion which is in relation with the aortic valves, in cases of rheumatic endocarditis. This fact is shown by the signs during life which indicate the mitral valve as the seat of disease, and by the larger proportion of instances in which mitral valvular lesions are observed in the cases in which organic affections of the heart are traceable to an attack of rheumatism. The anatomical characters of endocarditis artificially induced in inferior animals, in Dr. Richardson's experiments, as has been stated, were mostly limited to the auriculo-ventricular valves.

What is the nature of the pathological relation existing between endocarditis and acute rheumatism? The remarks in connection with a similar question as applied to pericarditis are here equally applicable. The endocardial inflammation is not developed as a metastasis of the articular affection. The former proceeds from the same morbid condition which determines the latter. Both are effects of a common internal cause. The affection of the joints is not lessened by the occurrence of endocarditis. Nor does the affection of the joints always precede the development of the endocardial inflammation. The latter occasionally takes precedence. Clinical observation appears to show that the liability to endocarditis is in proportion to the acuteness of the rheumatism; yet it is to be borne in mind that the mildest cases of rheumatism are not exempt from this liability. Endocarditis may be developed at any period during the career of acute rheumatism; but the statistics of Dr. Fuller go to show that the liability is greatest between the

sixth and twentieth days of the disease. The influence of youth in the development of rheumatic endocarditis does not appear to be so marked as with respect to pericarditis.

Endocarditis, as well as pericarditis, is one of the secondary inflammations liable to become developed in connection with Bright's disease. Of 39 fatal cases of recent endocarditis analyzed with reference to this point by Dr. T. K. Chambers,[1] it was referable to uræmia from Bright's disease in 12. In the same collection of cases, the affection was connected with acute rheumatism in only 9. These figures show a larger proportion of *fatal* cases from uræmic than from rheumatic endocarditis. The same, it has been seen, is also true with regard to pericarditis. The fact is owing to the greater fatality of endocarditis, as well as pericarditis, when associated with Bright's disease. The latter affection, existing to an extent to induce inflammation of any of the important organs of the body, generally proves fatal. On the other hand, acute rheumatism complicated with cardiac disease, very rarely ends fatally. The difference in fatality between the two affections thus accounts for the preponderance of fatal cases of endocarditis with Bright's disease, while the proportion of instances in which endocarditis is associated with acute rheumatism is vastly greater.

As regards the pathological relation existing between endocarditis and Bright's disease, the remarks made with respect to pericarditis are equally applicable. It is most consistent with our present knowledge to attribute the development of the former, as well as the latter, to the accumulation of urinary principles in the blood. The analogy of structure between the endocardium and serous membranes, explains the liability of the former to become inflamed under the same conditions which occasion inflammation of the latter.

It is stated that in non-rheumatic endocarditis, the aortic valves are more likely to be the seat of inflammation than the mitral, the reverse being true, as has been seen, of rheumatic endocarditis.[2] But in a pretty large proportion of instances, the endocardium in both situations is affected, whatever may be the pathological connection of the disease.

Endocarditis and pericarditis, as already stated, are frequently associated. This combination existed in 38 of the 204 cases analyzed by Dr. Fuller. Clinical observation shows that either

[1] Op. cit.

[2] Bellingham, op. cit., part ii. p. 348.

affection may take precedence of the other in point of time. Pericarditis, in fact, rarely exists without the coexistence of endocarditis. Hence, it may seem reasonable to infer that inflammation in either situation tends to develop it in the other, and, especially, that pericarditis leads to the development of endocarditis. It is doubtful, however, whether any causative relation exists between the two affections. When associated, they are probably effects of a common pathological condition. This condition, in the great majority of cases, is either rheumatic poisoning, or uræmia, but much oftener the former than the latter, clinical experience showing that endo-pericarditis is more frequently associated with rheumatism than with Bright's disease.

Endocarditis is sometimes associated with inflammation of the pulmonary structures—pleurisy or pneumonia. It is less frequently associated with these affections than pericarditis. The difference, as regards rheumatic endocarditis and pericarditis, is shown by the following statistics by Dr. Fuller:[1] Pulmonary inflammation, of some kind, existed in only 8 of 80 cases (a ratio of 1 to 10) of acute rheumatism complicated with endocarditis. It existed in 7 of 12 cases (1 to 1.7) of acute rheumatism complicated with pericarditis; and in 19 of 27 cases (1 to 1.4) in which endo-pericarditis was a complication. Pulmonary inflammation, however, exists oftener in cases of rheumatism complicated with endocarditis, than in rheumatic cases devoid of any cardiac complication. Thus Dr. Fuller found it to occur in only 7 of 127 cases in which rheumatism was uncomplicated with disease of the heart. This fact may seem to show that pulmonary inflammation exerts some influence, although feeble, in determining the occurrence of endocarditis. It is, however, more rational to conclude, in view of all the facts, that there does not exist any pathological relation between pneumonia or pleurisy and endocarditis when they are associated, but that both may proceed from a common cause. The occurrence of pulmonary inflammation in a proportion of cases somewhat larger when rheumatism is complicated with endocarditis, than when this complication does not exist, may arise from a greater intensity of the blood poisoning. If any pathological relation exists, it is perhaps as rational to suppose that the pulmonary inflammation is dependent on the cardiac affection, as that the former is involved in the causation of the latter.

[1] Op. cit.

Endocarditis is occasionally developed in connection with the eruptive and continued fevers, and with the morbid condition considered as pyæmia. Its occurrence in these connections is rare. It may also be produced by injuries of the chest; but cases of traumatic endocarditis must be extremely infrequent.

It is evident from the foregoing account of the pathological relations of endocarditis, that, exclusive of its connection with acute rheumatism and Bright's disease, it is, practically, not of much importance, since it so seldom comes under the observation of the medical practitioner. But, as before remarked, its occurrence as an idiopathic affection, especially in early life, may not be quite as infrequent as is generally supposed. The occasional instances of valvular lesions in children who have not had acute rheumatism, is a ground for suspecting that it occurs oftener than it is recognized. As a complication of rheumatism and Bright's disease, it possesses very great importance, in view of the frequency of its occurrence, and its remote evils. Its importance, in a certain point of view, is greater as a complication of rheumatism than of Bright's disease, for in the latter association it is generally combined with pericarditis, and it is developed under circumstances which offer small encouragement to expect recovery. On the other hand, in connection with rheumatism, the immediate danger is slight, and there is ground for hope that by appropriate management remote evils may be mitigated if not prevented.

The experiments of Dr. Richardson, to which reference has been made, are of interest and value in their bearing on the causation of endocarditis. After injecting into the peritoneal cavity of the dog a solution of lactic acid containing ten per cent. of the acid (an operation almost painless) the liquid is soon absorbed, and in about twelve hours the symptoms and physical signs denote the development of endocarditis. The morbid appearances observed in different stages of the endocardial inflammation have been stated. These appearances are mostly confined to the right side of the heart, and are especially seated in the tricuspid valve and orifice. Dr. Richardson attributes the inflammation to the local action of the lactic acid, which he supposes to act on the right side of the heart, because, being absorbed by the veins, "it comes into contact with the inner surface of the right side of the heart first; in the pulmonic circuit, it undergoes some loss, and so entering the left cavity is less active in its effects. In other words, in so far as the heart is concerned, the poison is derived from the systemic circuit, and is

lost in the pulmonic circuit." He regards his experiments as proving, synthetically, that rheumatic endocarditis is produced by a similar agent. Analysis furnishes corroborative evidence by showing the acidity of the excreta in acute rheumatism, especially the perspiration. But in rheumatism the endocarditis is seated in the left, not in the right cavities of the heart. To account for this Dr. Richardson supposes that the poison in rheumatic endocarditis is a product of respiration, and is contained in the arterial blood. "Hence, it comes in contact, first, with the inner surface of the left side of the heart; while, in the systemic circuit, it undergoes loss or combination, so that the blood returning by the veins is not poisoned, and the right side of the heart escapes." That the inflammation is produced by the direct contact of a poisonous agent, when artificially induced, and in rheumatic endocarditis, Dr. Richardson considers as proved by the limitation of the inflammation to one side of the heart, for if a blood-poison were to produce its effect through the nutritive vessels of the part, it would seem that the two sides of the heart should be equally affected, inasmuch as both are supplied from a common source with the same blood. The fact that during fœtal life, when the lungs do not fulfil the office of respiration, the right side of the heart is liable to endocarditis corroborates the views of Dr. Richardson.

Endocarditis may give rise to immediate pathological results which are important. Among these are emboli, consisting of detached masses of fibrin or lymph, or both, of greater or less size, which, propelled with the current of blood into the arteries, are at length arrested in their course in trunks too small to permit their farther progress, giving rise to arterial obstruction and diminished supply of blood to certain parts. This subject has already been considered in connection with valvular lesions, Chapter III., to which the reader is referred.[1] It is sufficient to say that the production of emboli is an accidental event which may occur during the progress of endocarditis, as well as at a later period when lesions of the valves have taken place as a remote effect of endocardial inflammation. Their occurrence, however, is more frequent in connection with valvular lesions. The phenomena which are symptomatic of embolic obstruction, such as apoplectic or epileptiform seizures, paralysis, etc., rarely enter into the clinical history of cases of endocarditis. But the liability to their occurrence, and

[1] *Vide* page 151.

the explanation, at least in certain instances, should be borne in mind.

The solid deposits in cases of endocarditis, viz., fibrin and lymph, are, to a greater or less extent, disintegrated by the blood-currents and carried into the circulation, either in solution or suspended in the form of minute particles. According to the observations of Dr. John Taylor,[1] the comminuted solid deposits, transported to different organs, and becoming arrested in the capillary vessels, may give rise to vascular obstruction and secondary inflammation in these organs. The kidneys and spleen are most likely to be the seat of disease thus induced. These effects are primarily mechanical; but it is highly probable that morbid changes in the blood itself are sometimes induced by the admixture of the liquid products of endocardial inflammation. It can hardly be otherwise, if, as is not improbable, purulent matter is occasionally formed on excoriated or ulcerated surfaces which are in some instances observed after death in cases of endocarditis. Our knowledge of these effects, as derived from clinical facts, however, is as yet too meagre to warrant any important conclusions. The fact that rheumatic endocarditis very rarely ends fatally, and rarely presents symptoms which denote purulent infection of the blood, goes to show the benignity, in most instances, of the liquid or soluble products of endocarditis.

The formation of coagula in the cavities of the heart belongs among the immediate pathological effects of endocarditis. The contact of the blood with the inflamed membrane, and the commingling of the liquid products of endocardial inflammation, have been supposed to induce coagulation, giving rise to the ante-mortem clots which were called by the older writers polypi of the heart. As an effect purely of endocarditis, this must be extremely rare, in view of the fact already repeatedly stated, viz., that endocarditis is fatal in but a very small proportion of cases. It is probable that when this event does occur during the progress of endocarditis, other conditions are involved which are more concerned in the occurrence of the event than the endocardial inflammation, such as cardiac enlargement, weakness of the heart from any cause, and a state of the blood which renders it prone to coagulation. Clinical observation fails in furnishing evidence of the formation of coagula during the progress of endocarditis as often as the writings of Bouillaud and some others would lead the observer to expect; but that

[1] Walshe, op. cit., 2d English edition, p. 610.

a certain amount of agency in the production of this event is derived from the disease, is not to be denied.

Symptoms of Endocarditis.

The symptoms of endocarditis are less distinctive than those of pericarditis. Occurring, generally, in connection with acute rheumatism, its symptomatic phenomena are merged in those of the latter affection. Hence, although its occurrence as a complication of rheumatism is so frequent, even the existence of such a disease has been known only within a few years past. In a large proportion of cases there are no symptoms which attract attention to the heart as the seat of any disease. Examination, however, with a view to determine the presence, or otherwise, of phenomena which point to endocarditis, may elicit symptoms which are of importance in the diagnosis. These symptoms consist of pain referable to the heart, symptomatic fever, and excited action of the organ, or palpitation. Symptoms arising from obstruction to the passage of blood through the orifices of the heart do not belong, properly, to the symptomatology of endocarditis, but are due either to lesions resulting from endocardial inflammation, or to accidental events, such as the formation of coagula. These symptoms have been considered in preceding chapters.

Pain is very rarely a prominent symptom, and it is often wanting. When present, it is not easy to refer it to the endocardium, except by taking into account other symptoms and the physical signs. The pain is not acute or lancinating, but dull, obtuse, or burning. A sense of uneasiness, hardly amounting to pain, is sometimes referred to the præcordia. The suffering from the affection of the joints is usually so much greater than the pain arising from rheumatic endocarditis, that the patient will not be likely to speak of the latter until interrogated with respect to it. If præcordial pain be marked in cases of rheumatism, there is reason to suspect not alone endocarditis, but pericarditis or pleurisy, and these affections are to be excluded by the absence of other symptoms and signs before concluding that the pain is due exclusively to endocardial inflammation.

It is probable that inflammation of the endocardium alone would generally give rise to more or less febrile movement. But in cases of rheumatic endocarditis it is difficult to say how much of the

febrile movement is symptomatic of the cardiac inflammation. Taken alone, this symptom is in no wise distinctive. If febrile movement be suddenly developed or increased when it is not referable to a fresh attack of any of the joints, or to inflammation seated elsewhere, it is fair to attribute it to the occurrence of endocarditis, if other symptoms and signs indicate the existence of this affection. Febrile movement, under the circumstances just stated, should excite suspicion of the occurrence of endocarditis, and lead to careful examination with reference to other symptoms and signs.

Endocardial inflammation may excite the muscular action of the heart, inducing a species of palpitation. Of this the patient may be conscious and make complaint; and it is apparent to the hand placed over the præcordia, and also by the pulse. The action of the heart may be irregular, as well as unduly excited. The force of the pulse is observed in some instances not to correspond with the activity of the heart, as shown by the impulse felt in the præcordia. These symptoms, occurring in the course of acute rheumatism, should lead to the suspicion of cardiac disease, which may prove to be endocarditis. Like the other symptoms, these alone are of little value as distinctive of the disease, but, taken in connection with other symptoms and signs, they have considerable significance.

The symptoms which have been mentioned derive their importance, as indicative of endocarditis, chiefly from their occurrence in the course of acute rheumatism. The clinical history of idiopathic endocarditis, based on analyses of recorded cases of the disease, is yet to be written. The information obtained by physical exploration is more important, and to this, attention will now be directed.

Physical Signs of Endocarditis.

Increased extent and degree of dulness on percussion, due to tumefaction of the heart and accumulation of blood within its cavities, is considered by Bouillaud and others as a physical sign of endocarditis. Assuming that these conditions are incident to the disease, it may fairly be doubted whether the cardiac enlargement often, if ever, much exceeds the limit of healthy variations; and if the size of the heart be found to be abnormal, it is impossible to say that it is owing to an existing endocarditis, unless it have been ascertained by previous examinations that, prior to the

present attack, there was no enlargement of the heart. If, by successive explorations from day to day, it be ascertained that the heart becomes enlarged, as it were, under the eyes of the observer, and it be clear that pericarditis does not coexist, the increased size may be attributed to endocardial inflammation, provided other signs and the symptoms are sufficient for the diagnosis. How far the size of the heart undergoes alterations during the progress of endocarditis I am unable to say from my own observations; but it is evident that percussion cannot afford very important information with reference to the diagnosis of this disease, except in a negative point of view, *i. e.*, by aiding in the exclusion of other cardiac affections, more especially pericarditis. In this point of view it is of much importance.

Palpation and inspection furnish evidence of excited action of the heart. The impulse is seen and felt to be more violent than in health, or out of proportion to the amount of febrile movement which exists. These signs, however, are present in but a certain proportion of cases, and may continue only during the early part of the disease. Moreover, abnormal activity of the heart, giving rise to increased force and extent of impulse, is sufficiently common in cases of functional disorder, irrespective of endocarditis. The signs furnished by these methods of exploration, are, therefore, of little value except as associated with other evidence of endocardial inflammation.

The only positive proof of the existence of endocarditis, is derived from auscultation, and consists mainly in the presence of an endocardial murmur. Clinical experience has established the fact that, as a rule, a murmur accompanies inflammation of the endocardium. This murmur is usually soft, having the character of a bellows-sound, and is systolic, *i. e.*, accompanies the first or systolic sound of the heart. It is not, however, developed always, and perhaps but rarely, at the commencement of the inflammation. A certain period elapses before this sign is discovered, and this period probably varies in different cases. It is not easy to determine the interval in many instances, since the existence of endocarditis is not positively determinable prior to the production of a bellows-murmur. An approximation to correctness of observation with respect to this point, is obtained by ascertaining the duration of the symptoms which point to cardiac disease, anterior to the development of the murmur. The average duration of inflammation before murmur occurs, is yet to be determined. Dr. Richardson found that a

murmur invariably followed the symptoms of endocarditis artificially induced in inferior animals, the interval varying in his different experiments. We are not, however, warranted in stating that endocarditis never exists without a murmur being produced, sooner or later, during the progress of the disease. The exceptions to the rule are probably quite rare, but this is a point to be settled more definitely by future researches.

It is needless to remark that an endocardial murmur is not, in itself, evidence of existing endocarditis. Murmur occurs in connection with valvular lesions which have taken place as remote effects of inflammation, or as results of other causes. It occurs in consequence of blood-changes, independently of an inflammatory affection or any organic disease of the heart. The inquiry then arises, what are the circumstances which, taken in connection with the presence of a murmur, render it a diagnostic criterion of endocarditis? This inquiry, from its obvious practical bearing, claims careful attention.

The development of a murmur in the course of acute rheumatism, in conjunction with symptoms denoting cardiac inflammation, renders it almost certain that endocarditis has occurred. If, after the lapse of several days, an endocardial murmur be detected, which previous explorations, made with sufficient care, have failed to discover, the practitioner should conclude that it is a sign of endocarditis. This conclusion is rendered more positive, if increased febrile movement, excited action of the heart, or pain in the præcordia, are observed to precede or accompany the development of the murmur. But it will happen not infrequently that a murmur is present when the patient first comes under observation. This is the case especially in hospital practice, patients being admitted after rheumatism has continued already for a greater or less period. It happens also in private practice, since endocarditis may occur at the very commencement of an attack of rheumatism, and may even precede it. The difficulty, in these instances, is to determine that the murmur has been recently developed. It may have existed prior to the attack of rheumatism, being dependent on some organic mischief, or on inorganic morbid conditions. There is strong ground for suspecting that the murmur pre-existed, if the patient have had rheumatism before. This difficulty is sometimes insuperable; but with reference to it, several points are to be considered. A murmur due to endocarditis is generally referable to the mitral orifice; in other words, it is either limited to, or heard with greatest

intensity over, or near the point of the apex-beat of the heart. It has been seen that in rheumatic endocarditis, the inflammation is seated especially at the mitral valve, and clinical observation shows that, in most instances, the murmur emanates from this situation. The fact of the murmur being mitral, shows that it is not inorganic, since an inorganic murmur, in the vast majority of cases, if not invariably, is produced at the arterial orifices. If, however, the murmur in question be aortic, other circumstances are to be taken into account in determining that it is not inorganic. These circumstances have been considered in a preceding chapter.[1] Having determined that the murmur is not inorganic, the question then is, whether it be due to valvular lesions which have existed for a greater or less period, or whether it denote an existing endocarditis. Valvular lesions lead to enlargement of the heart. Now, if the heart be found to be enlarged, it is probable that the murmur proceeds from valvular lesions. Endocarditis, it is true, may occur, and is perhaps more likely to occur, in cases of rheumatism, when the heart is already affected with organic disease, but, under these circumstances, the murmur cannot be considered as a sign of endocarditis. On the other hand, if the heart be not enlarged, the chances are in favor of the murmur being due to endocarditis, especially if the symptoms render the existence of the latter probable. Another point relates to the murmur itself, assuming that it is referable to the mitral orifice. A murmur due to existing endocarditis is soft, usually not intense, and limited to a circumscribed space. Roughness and great intensity denote valvular lesions. Diffusion of the murmur over the left lateral and posterior surfaces of the chest, indicates lesions which permit free regurgitation. Diastolic murmurs are usually, if not always, due to valvular lesions; consequently, a systolic murmur cannot be considered as a sign of endocarditis, if a diastolic murmur be also present. The previous occurrence of rheumatism is to be taken into account. Other things being equal, the chances that a murmur proceeds from endocardial inflammation are more if rheumatism have not occurred previously. Attention to these points will enable the practitioner to decide, not always with positiveness, but with an approximation toward certainty, whether a murmur be, or be not significant of endocarditis.

A murmur developed by endocarditis generally continues not

[1] *Vide* page 202.

only during the continuance of the disease, but ever afterward. There are exceptions to this rule. I have known, in several instances, a mitral murmur to disappear entirely after recovery from rheumatic endocarditis, when, during the progress of the disease, and for some time afterward, it had been well marked and constant. This is to be accounted for by supposing that the swelling of the valves diminishes, the deposits of lymph and fibrin are gradually disintegrated and washed away, and the endocardial surface is rendered smooth by the currents of blood, so that the physical conditions for the production of murmur are no longer present. But in the majority of cases the murmur not only persists, but increases rather than diminishes in intensity, in proportion as valvular lesions become more and more declared. It may continue, however, for many years without any notable alteration.

What are the physical conditions incident to endocarditis which give rise to a murmur? It is probably due to roughness of the endocardial membrane covering the valves, produced by the deposits of lymph and fibrin. It has been conjectured that, in consequence of spasmodic action of the papillary muscles, the mitral valve fails to fulfil its function, and regurgitation takes place. This is inconsistent with the constancy of the murmur and its persistence after recovery from the endocarditis. It is not necessary to assume the occurrence of regurgitation in order to account for a mitral systolic murmur. The murmur is produced in the ventricle, in other words, it is intra-ventricular, although emanating from the mitral orifice. The presence of the solid products of inflammation is sufficiently adequate to explain the occurrence of the soft, feeble and circumscribed murmur which characterizes endocarditis.[1]

The heart-sounds may present certain abnormal modifications in endocarditis. Reduplications are sometimes observed. One or both of the sounds, the first sound more especially, may be less distinct than in health. The first sound may be wanting. Dr. Richardson, in his experiments, found that the first sound frequently disappeared for some time before a murmur was developed. It is

[1] I should add that I have observed a mitral systolic murmur to disappear before the termination of rheumatism. I have even noted the existence of this murmur during one day only, careful auscultation failing to discover any the day previous and subsequently. But in this case it seems to me more reasonable to attribute the production of the murmur to the deposit of fibrin or lymph which was soon washed away by the currents of blood, than to spasmodic action of the papillary muscles.

not difficult to conceive of this in view of the great swelling of the auriculo-ventricular valves which he observed when the animals were killed during the early stage of the inflammation—the segments resembling an injected uvula, and lying so close to each other that, when the heart was contracting, they must have cushioned against each other, fulfilling their office of preventing regurgitation without tension or movement. Theoretically, it would be expected that the mitral valvular element of the first sound should be lessened or extinguished, the tricuspid valvular element remaining unimpaired. It is not improbable that this change may precede the development of a murmur, and thus be of value as an earlier physical sign than the latter. With respect to this point I cannot speak from clinical observation.

Diagnosis of Endocarditis.

The diagnosis of endocarditis rests on physical evidence. It is impossible to determine the existence of the disease by means of the symptoms alone; it is therefore necessarily overlooked by those who do not employ physical exploration. The evidence consists in the development of an endocardial murmur, in connection with symptoms which corroborate its significance. In a certain proportion of cases, the diagnosis may be made with positiveness. When the development of a murmur is a matter of observation, under circumstances which render the occurrence of the disease probable, there is no room for doubt. The diagnosis is less easy, often difficult, and sometimes impossible, in cases in which the newness of a murmur is to be determined, not by observation, but inferentially. When this is the case, the practitioner is liable to err, on the one hand, in basing his diagnosis on the presence of a murmur which is not newly developed, and, on the other hand, in attributing a newly-developed murmur to other conditions than an existing inflammation.

In a patient who presents unequivocal evidence of valvular lesions, the diagnosis of endocarditis is extremely difficult, and often impossible. How is the practitioner to determine that murmurs, under these circumstances, are due to existing inflammation, and not to the valvular lesions? It is possible, if a case have been under observation previously, that certain changes in the situation and character of the murmur may be fairly attributable to super-

induced endocarditis, but this will happen in only a small proportion of instances. I have met with cases repeatedly in which murmurs connected with old valvular lesions have been considered as evidence of inflammation, and a course of treatment pursued which was highly prejudicial to the welfare of the patients. But the error, in these instances, proceeded from a very imperfect knowledge of the diseases of the heart. A question as to the existence of endocarditis in connection with long standing organic disease, can hardly arise except during an attack of acute rheumatism. Not infrequently, under these circumstances, the question does arise. A patient has had one or more previous attacks of rheumatism, which have led to organic disease of the heart. A fresh attack occurs. The symptoms and signs referable to the heart may be due wholly to the pre-existing organic disease, or they may, in part, proceed from new physical conditions incident to an existing endocardial inflammation. To determine positively with respect to this point, is certainly one of the most difficult problems in diagnosis. The problem, in fact, cannot be solved with positiveness. Cases of organic disease of heart, therefore, are to be excluded from the category of those in which a diagnosis is generally practicable.

Idiopathic endocarditis is certainly one of the rarest of diseases. But, as already stated, there is reason to believe that it occurs, especially in children, when it is not recognized. It would perhaps be discovered in some instances in which it is overlooked, if practitioners, in the first place, were more generally qualified to employ physical exploration, and, in the second place, if it were more the custom to auscultate the heart even when the symptoms do not point distinctly to disease of that organ. Is the diagnosis of idiopathic endocarditis practicable? An endocardial murmur developed under the observation of the practitioner, preceded and accompanied by pain or uneasiness in the præcordial region, febrile movement and excited action of the heart, other affections which might give rise to these symptoms being excluded, would warrant a positive diagnosis. It is, however, hardly to be expected that this combination of circumstances will often be presented in practice. The symptoms will not be so well declared, and at the first examination a murmur may be found, the previous duration of which is indeterminate. I confess that I have no knowledge of idiopathic endocarditis, derived from the clinical study of the disease, and older, as well as better observers have made the same confession. Dr. Stokes remarks with reference to this subject: "In

truth, we rarely meet with a case of simple, idiopathic endocarditis fit to be considered as a type of the signs and symptoms of the disease. Such a case, at least, has never occurred to me."[1]

Pericarditis is so frequently associated with endocarditis, that the existence of the former renders the coexistence of the latter highly probable. But in some instances of endo-pericarditis, an endocardial murmur may be, for a time, wanting, being obscured by the friction-sound, or the heart being too much weakened by the compression of liquid effusion to produce it. On the other hand, the pressure of liquid effusion and lymph on the large vessels within the pericardium has been supposed to give rise, in some instances, to murmur at the arterial orifices. Of course when pericarditis exists, symptoms referable to the heart are of no value as respects the diagnosis of endocarditis. The combination of inflammation of the lining and investing membrane of the heart is more serious than either affection singly; but happily, the diagnosis of endocarditis, under these circumstances, although it affects the prognosis, does not influence materially the treatment.

Prognosis in Cases of Endocarditis.

What is to be said under this head has been anticipated in the foregoing remarks. The prognosis, as regards immediate danger to life, and even the continuance of symptoms which occasion inconvenience, is favorable. Recovery in most instances appears to be complete. It is possible that the inflammation may become chronic and persist for an indefinite period. This may be suspected, if the patient complain of uneasiness in the præcordia, and the action of the heart continues unduly excited. But our knowledge of chronic endocarditis is not sufficient to furnish grounds for discriminating between it and valvular lesions resulting from changes which take place after inflammatory action has ceased. Hence, there is no advantage in treating of acute and chronic endocarditis separately. Certain contingent or accidental events, of a serious nature, to which reference has been already made, are liable to occur during the progress of endocarditis. These are, the formation of coagula; the detachment of masses of fibrin or lymph, constituting emboli; the admixture of disintegrated solid deposits, and

[1] Op. cit., Am. ed., p. 118.

purulent infection of the blood. Clinical observation, however, shows that in a large majority of cases of endocarditis, recovery takes place without serious accidents; the symptoms referable to the heart, if any were present, disappear, leaving the patient exposed to the evils arising from valvular lesions which may become developed at a period more or less remote.

Treatment of Endocarditis.

The objects of treatment in pericarditis and endocarditis are not in all respects similar. In pericarditis, the compression of the heart by the accumulation of liquid within the pericardial sac is a source of distress and danger. To prevent this accumulation, and promote its removal, are important therapeutical ends. In endocarditis, the action of the heart is free from all mechanical restraint. In pericarditis, the inflammation is more generally diffused, and a greater effect is produced upon the muscular walls, first by excitation, and afterwards by paralysis. In endocarditis, the inflammation is seated especially in the membrane connected with the valves and orifices, where it is not in contact with the muscular walls, and the latter are consequently affected in a less degree. In pericarditis, the aim of the practitioner is often to avert impending death. In endocarditis, there is little fear of a fatal result. But although the two affections are so dissimilar in many respects, the general principles of management are in a great measure alike applicable to both.

The therapeutical indications in the treatment of endocarditis relate mainly to the alterations to which the membrane is exposed, and to the products of inflammation. The objects are to lessen, as far as possible, the local effects of the inflammation, to aid in restoration from these effects, and thus protect the organ from the remote consequences arising from incurable and progressive unsoundness. These objects embrace measures having in view abatement of the intensity of the inflammation, abridging its duration, limiting the exudation of lymph and the precipitation of fibrin, and effecting the removal of solid deposits. The measures for these ends are those which were involved in the treatment of pericarditis, viz., bloodletting and other antiphlogistic measures, mercurialization, opium, sedatives, eliminatives, and counter-irritation.

In the employment of bloodletting, the practitioner is to be guided by the same indicating and contra-indicating circumstances

as in other inflammations. This remedy is indicated, and the extent to which it is to be carried is to be regulated by the apparent intensity of the inflammation, the state of the vascular system, the constitution of the patient, and its immediate effects. It is contra-indicated by weakness of the circulation, feebleness of constitution, anæmia, and when, upon trial, want of tolerance of the remedy is apparent. The indications are present in a certain proportion of the cases of idiopathic and rheumatic pericarditis, but rarely, if ever, when the disease is developed in connection with Bright's disease. The remarks with respect to this remedy in pericarditis are, in general, here applicable; but the danger incident to the injudicious employment of bloodletting is greater in pericarditis, in view of the tendency of the latter to induce weakness and paralysis of the heart. Aside from the effect of bloodletting in diminishing the intensity of inflammatory action, it may be useful by lessening the labor which the heart has to perform, and preventing the accumulation of blood within its cavities. Bloodletting in endocarditis, as in other inflammations, is to be employed only during the early part of the disease. It is not called for by the disease *per se*, but by the circumstances attendant on the disease. General or local bloodletting may be employed, the latter when it is not desired to abstract a large quantity of blood, or to withdraw it rapidly. In most instances the indications for bloodletting will be fulfilled by leeching or cupping.

The measures which, in conjunction with bloodletting, constitute the antiphlogistic treatment, are purgation and low diet. Purgative remedies may be employed as a means of depletion when circumstances contra-indicate bloodletting. The saline purgatives are best suited for this purpose. Depletion is also effected indirectly by limiting the supply of nutriment. These measures, as well as bloodletting, are appropriate to the early period of the inflammation. It is, to say the least, useless to continue them after the inflammation has continued sufficiently long to produce all the immediate local effects to which it is likely to give rise. After the lapse of a few days from the date of the attack, they are not indicated more than, for example, in the second or exudation stage of pneumonia.

Mercury is generally regarded as a highly important remedy in endocarditis, from its supposed power in controlling the processes of inflammation, restraining exudation, diminishing the tendency to the deposit of fibrin, and promoting absorption of the products

of inflammation. Although its efficacy in these several ways has doubtless been much exaggerated, we are not authorized to say that it is in no degree useful; and the evils or inconveniences of careful mercurialization are trivial in comparison with even a small amount of usefulness. It is probable that all the benefit to be obtained from this remedial agent is secured by pushing it cautiously to the extent of producing slight ptyalism. It should not be carried beyond this effect, nor continued after this effect is induced. It is needless to add that mercurialization is improper in this disease under the same circumstances which contra-indicate it in pericarditis or other inflammations.

The pain in endocarditis is rarely sufficient to call for opiates. But it is fair to infer from the apparent usefulness of opium in inflammations affecting analogous structures, that it is a useful remedy in this disease. It is also indicated, as a sedative, when the action of the heart is unduly excited.

Other sedatives, such as digitalis, antimony in small doses, or the veratrum viride, may sometimes be useful in reducing the excited action of the heart. They should not be carried to the extent of weakening the heart's action, for, although there is not so much immediate danger from this effect as in pericarditis, it must be unfavorable by preventing the completeness of the ventricular contractions and favoring the accumulation of blood in the cavities of the heart.

Eliminative remedies are indicated in endocarditis on precisely the same grounds and to the same extent as in pericarditis, when the disease occurs in connection with acute rheumatism or with Bright's disease. The treatment in the latter affections which is most effective in removing from the blood the poisonous principles giving rise to local inflammations will prove most effectual in preventing the development and the persistence of endocarditis. The remarks under this head in connection with pericarditis are equally pertinent to the present subject, and need not be repeated. Certain facts observed by Dr. Richardson in his experiments are interesting with reference to the effect of eliminative remedies. In about twelve hours after the injection of the lactic acid solution into the peritoneum, when the symptoms denoting the commencement of endocarditis became developed, if the animal was freely purged or passed a large quantity of urine, the symptoms all subsided, and renewal of the injection was necessary in order to sustain the effect.

Counter-irritants, viz., sinapisms, blisters, pustulation with croton

oil, and stimulating liniments, are indicated in the treatment of endocarditis as in pericarditis, the only difference between the two affections as regards the application of these remedies consisting in the fact that in pericarditis the absorption of liquid effusion may be promoted by vesication, while in endocarditis this is not an object of treatment.

Regarding the treatment of endocarditis from another point of view, viz., with reference to the objects or indications which are presented during the progress of the disease, the measures which have been mentioned may be recapitulated, and some additional points relating to the management noticed.

Bearing in mind the frequent occurrence of the disease in the course of acute rheumatism, it is an indication to endeavor to prevent its development. Without discussing the treatment of rheumatism, it is sufficient to say that measures which eliminate the *materies morbi* from the blood are those which, rationally considered, must prove most efficient in the way of prophylaxis. These remarks are also applicable to the prevention of endocarditis in cases of Bright's disease.

At the commencement of endocarditis, and during the early part of the disease, it is an object of treatment to diminish the intensity of the inflammation. This object is important not on account of any immediate danger to life, however intense the inflammation, but in order to limit its local effects, more especially as regards the products of exudation. The means for accomplishing this object are essentially those which are regarded as useful at the onset and during the early stage of inflammation affecting analogous structures. They consist of local or general bloodletting in certain cases, saline purgatives, and low diet. In pursuing these measures, the practitioner is to be guided, not by the mere fact that endocarditis exists, but by the associated circumstances in individual cases, giving due consideration to those which may contra-indicate bloodletting and other modes of depletion. These measures are not to be employed or continued when the inflammation has existed for several days, the immediate local effects of the inflammatory process having then already taken place, so far as these are dependent on the intensity of the inflammation.

Assuming that mercury exerts any influence to limit inflammatory exudation, it is indicated at the commencement of the disease, and the special effects of this remedy should be induced as speedily as possible, discontinuing it so soon as these effects are produced.

The solid deposits incident to endocarditis having been seen to consist, in part, of coagulated fibrin derived from the blood contained within the cavities of the heart, and this effect being in a measure dependent on an excess of fibrin in the blood, especially when the disease occurs during the course of acute rheumatism, it is a rational indication to endeavor to diminish the quantity of fibrin (hyperinosis) by therapeutical measures. Mercury, and alkaline remedies have been supposed to fulfil this indication, but their efficiency cannot be considered as established. Physiological experiments which show the destruction of fibrin in the liver and kidneys, suggest the inquiry whether remedies exciting the functions of these organs may not be useful by reducing an abnormal proportion of fibrin in the blood.[1]

It is probable that more or less of the solid deposits which take place in endocarditis are removed by absorption. The deposits beneath the endocardial membrane can only be removed in this way. Those occurring on the free surface of the membrane, may be removed, partly or entirely, by the friction of the blood in motion. If it be possible to promote absorption of the products of inflammation by remedies, these are obviously indicated, inasmuch as the remote evils of the disease arise, in a great measure, from the permanence of the deposits. Mercury and the iodide of potassium are considered as useful in fulfilling this indication.

Pain in some cases, and more frequently excited action of the heart, call for opium. This remedy, there is reason to believe, is useful in this, as in other inflammations, not merely as a palliative of suffering and a sedative, but from a power of controlling, to some extent, the inflammatory processes. Other cardiac sedatives, such as digitalis, antimony, and the veratrum viride, are indicated by excited action of the heart, but they are to be employed with circumspection, so as not to weaken unduly the muscular power of the organ.

It is an important object of treatment to prevent the persistence of endocarditis after it has existed for several days, and its intensity is diminished. Counter-irritant applications are indicated for this object. It is probable that these exert more or less effect in hastening the complete disappearance of the inflammation.

[1] *Vide* Essay on the Rapidity and Extent of the Physical and Chemical Changes in the Interior of the Body. By Prof. John C. Dalton. Trans. New York Academy of Med., Vol. II. Part III.; and New York Monthly Review of Med. and Surg. Science, No. for Sept. 1859.

The indications during convalescence, and subsequently, are essentially the same as during and after recovery from other inflammations affecting important organs. Avoidance of causes which may reproduce the affection is important. The powers of the heart should not be unduly tasked by violent exercise, abuse of stimulants, or excesses of any kind. It may be doubted whether an amount of physical activity necessary to vigorous health, be unfavorable as regards the liability to organic disease. A restricted diet, habits of inactivity and other measures calculated to enfeeble the system, are more likely to hasten than postpone the development of structural lesions. It is injudicious to lead the patient to anticipate the occurrence of remote evils which he may escape, and against which, at all events, he cannot be forearmed by being forewarned. The moral effect of looking forward to organic disease of heart may prove unfavorable to a condition of mind and body which is not only conducive to present comfort, but affords, in some degree, a protection against the danger to be apprehended.

In concluding these remarks on the treatment of endocarditis, two or three practical points remain to be noticed. In a pretty large proportion of cases the inflammation is not intense; it is evidently subacute, at least as represented by the symptoms. These are so far from being prominent, that the disease is habitually overlooked by those who do not resort to physical exploration, and its occurrence was unknown prior to the application of auscultation to the study of cardiac affections. In these cases the expedieney of very active therapeutical interference is doubtful. Bloodletting and other reducing measures are of questionable propriety, and the tendency to employ heroic remedies, or to push them too far, in view of remote evils, is to be resisted. Here, as in other forms of disease, as much injury may be done by excessive as by insufficient treatment. Another point relates to the period when the inflammation has ceased, and, consequently, the indications for treatment having reference to inflammation are no longer present. It is not always easy to determine when this period arrives. But it is important to warn the practitioner against attaching undue importance to the continuance of an endocardial murmur. This will be likely to persist, although the inflammation does not continue, for an indefinite time, and generally ever afterwards. The persistence of the murmur is no proof of inflammation, and does not, of itself, indicate the need of therapeutical measures. The symptoms must be relied upon in determining the intensity of the inflammation

during the course of the disease, and the period of its cessation. The latter is declared by the disappearance of pain or uneasiness in the præcordia, absence of febrile movement, and quietude of the heart's action. Finally, the importance of not attributing to endocarditis the symptoms which may be associated with an endocardial murmur in cases of organic disease is to be enforced. I have met repeatedly with instances of valvular lesions of long standing, in which bloodletting, low diet, mercurialization, etc., had been employed with a view to combat existing inflammation. It is important to avoid this error, since, in a large proportion of the cases of organic disease of heart, these therapeutical measures are injurious.

MYOCARDITIS.

Inflammation of the muscular structure of the heart constitutes the affection called carditis or myocarditis. Treating of this affection so far as it is of interest and importance to the physician, in a practical point of view, a brief consideration will suffice, without any formal subdivision of the subject.

The muscular substance of the heart is the seat of inflammation much less frequently than the investing and lining membranes of the organ. But, according to Rokitansky, inflammation occurs in this situation oftener than is generally supposed. As occurring independently of pericarditis and endocarditis, myocarditis is extremely rare. A few cases only are on record. Either the pericardium or the endocardium, or both membranes, are implicated in the great majority of the instances in which the muscular tissue is found after death to present the evidences of inflammation. The inflammation, probably, in most instances, extends from the investing or lining membrane to the muscular substance; but the latter may be primarily affected. The inflammation is usually limited to certain portions of the heart, and it occurs much oftener in the left than in the right ventricle. It may be confined to the outer or inner layers of muscular fibres, or it may extend throughout the walls, and affect the *columnæ carneæ*. The septum is less liable to be affected than the ventricular walls.

If suppuration take place, pus is found either in small collections, forming abscesses, or infiltrated more or less throughout the mus-

cular walls. When abscesses exist, the surrounding parts present, at the same time, purulent infiltration. The formation of abscesses involves destruction of the muscular structure to a greater or less extent. They are usually formed in the left ventricle. In a case reported by Dr. Graves, a collection of two ounces of pus was found in the walls of this ventricle. The muscular substance in the parts infiltrated is livid, softened, and more or less disintegrated. Abscesses may discharge their contents, by perforation, into the pericardial sac, giving rise to acute pericarditis, if the latter be not already present. Or they may evacuate into the ventricular cavity, in this case giving rise to purulent infection of the blood. In either case a fatal result is inevitable. An abscess formed in the ventricular septum has been known to lead to communication between the two ventricles.

Another, and, according to Rokitansky, a more frequent, termination of myocarditis is induration of the walls of the heart from the deposit of lymph and the formation of fibroid tissue. This termination involves weakness and atrophy of the muscular substance.

An ulterior result of myocarditis is aneurismal dilatation of the walls of the heart. These are fully described by Rokitansky, and have been referred to in a previous chapter. Rupture of the heart is an event in some instances incidental to inflammation of the cardiac substance.

Clinically considered, myocarditis is almost invariably associated with pericarditis, endocarditis, or endo-pericarditis, and its existence is not determinable during life. It may sometimes be suspected when the gravity of the cardiac symptoms is out of proportion to the apparent amount of endocardial or pericardial inflammation. But this statement is indefinite. There are no symptoms nor signs which warrant a diagnosis even approximating to positiveness. This remark will apply also to the very rare instances in which inflammation is limited to the muscular substance, the lining and investing membranes remaining unaffected. There would, therefore, be no advantage, practically, in dwelling on the subject. It is obvious that in proportion as myocarditis is added to endocarditis and pericarditis, singly or conjoined, the symptoms referable to the heart will denote increased gravity of cardiac disease, and the immediate danger is augmented. The patient is also exposed to certain accidents which have been mentioned, viz., rupture, aneurismal

dilatation, perforation of the inter-ventricular septum, and purulent infection of the blood. The discovery of these, during life, does not come within the reach of diagnosis.

As regards the treatment both of myocarditis and its accidents, the therapeutical measures which are likely to prove of any avail are perhaps indicated by the symptoms as clearly as if the diagnosis were practicable.

CHAPTER IX.

FUNCTIONAL DISORDER OF THE HEART.

Definition, and the different forms of disorder—Pathological relations and causation of functional disorder—Association with plethora, anæmia, various derangements of the nervous system, dyspepsia, gout, etc.—Symptoms of functional disorder—Physical signs furnished by percussion, palpation, and auscultation—Diagnosis of functional disorder—Prognosis—Treatment.

By functional disorder of the heart is meant disturbed action occurring independently of either inflammatory or organic affections. These affections usually involve more or less functional disorder, but the latter often occurs when the former are not present, being purely dynamic, or pertaining exclusively to the vital properties of the organ. In most instances the disturbed action of the heart is evidently due to morbid conditions seated elsewhere. It is usually symptomatic of either blood-changes, or affections of the nervous system, and, not infrequently, of both conjoined. These morbid conditions, although they are independent of inflammation and structural lesions, may, nevertheless, be associated with the latter. It is a fact important to be borne in mind, that disordered function of the heart, in certain cases of inflammatory, and, more especially, organic affections, involves the same morbid conditions which often exist independently of these affections. This is a practical point which will be again referred to. The subject of functional disorder of the heart is of great importance in a practical point of view, on account of the frequency of its occurrence, the anxiety which it occasions, and the liability of confounding it with organic disease. Of the persons who make complaint of symptoms referable to the heart, a large majority suffer from functional disorder only. But the discrimination of functional from organic affections can only be made by one who is thoroughly acquainted with the subject. The immense importance of discriminating correctly is obvious, when it is considered that structural lesions involve more or less danger, while disorder of function, although

often in a high degree distressing, very rarely, if ever, proves serious.

Functional disorder of the heart is not always identical, but presents certain varieties in different cases. In the mildest form of disorder, the action of the heart is simply increased unduly by transient exciting causes, such as mental emotions, muscular exercise, ingestion of food or stimulants, etc. The organ is morbidly excitable, but its action is not disturbed to an extent to occasion great inconvenience or annoyance.

Persisting inordinate action is another form of disorder. I have met with several instances in which the heart acted regularly, but with abnormal rapidity and force, irrespective of any exciting causes, the excited action continuing constantly for days, weeks, and even months. The pulse in these instances was uniformly frequent—from 110 to 120 per minute. The patients were conscious of an undue force of impulse and intensity of the heart-sounds; it was difficult for them to withdraw their attention from the action of the heart, and to overcome a conviction of the existence of organic disease.[1] This is not a frequent form of disorder. It is observed in females much oftener than in males, and it is sometimes associated with enlargement of the thyroid body and protuberance of the eyeballs.

As commonly presented in practice, functional disorder occurs in paroxysms, and the rhythm of the heart's action is disturbed. Either with or without an obvious exciting cause, the patient is conscious of violent beating of the heart. The movements of the organ, in severe cases, are tumultuous and extremely irregular; the systolic contractions at one instant following in rapid succession, at another instant more slowly, and intermittency occurring more or less frequently. The patient is painfully conscious, not only of the morbid intensity of the action, but of the rhythmical disturbance. Absolute repose is necessary. A feeling of impending death is experienced. Great anxiety and apprehension usually accompany the paroxysms, especially at first. The terror of the patient, in fact, not infrequently enhances considerably the disorder. The paroxysms may continue for a few moments only, or for several hours. Their severity varies much in different cases. In mild cases, as when they occur in connection with hysteria, the sense of disturbance consists in fluttering movements referred to the præcordia. As regards the recurrence of the paroxysms, cases vary

[1] Case of Mrs. M., Private Records, vol. ix. p. 424.

greatly. They may recur at short intervals, being easily provoked by various exciting causes, and occurring spontaneously; or they may take place at periods more or less remote. The disturbance of rhythm in these paroxysms is sometimes so great that, to quote the language of personification used by Bouillaud, the heart seems to be affected with a species of *insanity* (*une folie véritable*), beating at random, instead of with that regular, definite purpose which seems almost to involve a motive in its healthy action.

Another species of paroxysm is characterized by irregularity and intermissions, without increased force of the heart's action, but, on the contrary, the action of the heart may be quite feeble. I have observed these paroxysms to occur in a person liable to functional disorder of the heart, especially on exposure to cold, during fatigue from muscular exertion, and when the habitual time of taking food was delayed. This variety is even more distressing than that in which the paroxysms are characterized by violence of the heart's action. The feeling of impending death is rendered more vivid by a tendency to syncope.

Another paroxysmal variety consists in a sudden momentary disturbance, which is either an intermission or apparently a trembling movement of the heart, occurring at rare intervals, or more or less frequently. These paroxysms, until the mind becomes accustomed to them, inspire great terror. The patient feels, after they have passed, as if he had just escaped sudden death, and this feeling often causes the heart to beat rapidly after the paroxysms have ceased. After a time, patients become habituated to their occurrence, and they occasion much less apprehension. I have met repeatedly with persons who have been subject to them for a great number of years.

These are the varied forms under which functional disorder has presented itself in my own clinical experience. Different varieties, however, are frequently associated in the same case. All the forms are commonly embraced under the head of palpitation. They are also called, in distinction from inflammations and structural lesions, inorganic affections of the heart. As before remarked, functional disorder, in general, depends on morbid conditions seated elsewhere than in the heart. These causative conditions are by no means the same in all cases. A correct appreciation of the pathological relations of the disturbed cardiac action in individual cases is essential with reference to appropriate treatment. To these relations attention will now be directed.

Pathological Relations and Causation of Functional Disorder of the Heart.

Of the different morbid conditions on which functional disorder of the heart is dependent, clinical observation shows the most important to be plethora, anæmia, derangement of the nervous system induced by various causes, dyspepsia, and the gouty diathesis.

In the condition known as plethora, in which the blood is abnormally rich in red globules, and, perhaps, in excess as regards quantity, the heart appears to be overtasked and over-stimulated, and becomes, in consequence, morbidly irritable. Functional disorder, thus induced, is characterized by violence of action, with or without disturbance of rhythm. Palpitation may be the first symptom of the plethoric condition, which awakens the anxiety of the patient respecting the state of his health. His attention is usually at once concentrated on the heart, and he is fearful of organic disease. Cases which fall under this head are presented in persons who have altered their mode of life, exchanging habits of physical activity for sedentary pursuits or luxurious leisure. Students coming from the farm or workshop, men of business retiring to live in ease, and all who, in addition to indolence, cultivate the pleasures of the table, are liable, among other evils, to suffer from functional disorder of the heart incident to plethora. These cases are to be discriminated from others in which the pathological relations are quite different, with reference to the proper treatment.

Cases of functional disorder are much oftener met with in connection with a condition the opposite of plethora, viz., anæmia. It is rare for well-marked anæmia to exist without more or less disturbance of the heart's action. Cases belonging to this class occur vastly oftener among females than males, anæmia being as infrequent with the latter, as it is common with the former. Anæmia being produced by hemorrhages, leucorrhœa, frequent childbearing, prolonged lactation, etc., the functional disorder of the heart will, of course, in individual cases, be referable to one or more of these ulterior pathological relations, the anæmia, however, being the intervening causative condition. But the degree of disorder is not always proportionate to the anæmia, being sometimes slight when the anæmic state is marked, and, conversely, severe in some cases in which the latter is scarcely appreciable. Anæmia giving rise to

a multitude of morbid effects, in addition to disturbance of the heart's action, more or less of these are associated with the latter. Patients with functional disorder dependent on anæmia will be likely to present as symptoms, either coexisting or developed in succession, coldness of the extremities, spinal irritation, cephalalgia, neuralgic affections in different situations, depression of spirits, etc. Of all the associated morbid effects of anæmia, the cardiac disorder often occasions the most annoyance and anxiety. The fear of organic disease and sudden death is added to the distress which belongs intrinsically to the disorder. In cases of marked anæmia, patients are frequently supposed to labor under organic disease of the heart by those who trust exclusively to symptomatic phenomena in diagnosis. The symptoms, in fact, sometimes point strongly to the existence of organic disease. Not infrequently, palpitation is excited by the slightest exertion; dyspnœa is experienced; pain or uneasiness is referred to the præcordia; the countenance is morbid, and, occasionally, the hydræmic condition of the blood leads to œdema and anasarca. I have met with several instances in which all the symptoms of advanced organic disease of the heart were simulated by the morbid effects of anæmia induced by prolonged lactation and other causes. The importance of a correct diagnosis, as regards the prognosis and treatment, in these cases, is truly immense.

Derangement of the nervous system is doubtless the immediate cause of cardiac disorder in cases of anæmia. The morbid condition of the blood leads to disturbance of the heart's action through the intervention of the nervous system. But the latter may be deranged and functional disorder of the heart produced by various causes, irrespective of anæmia. Cases of hysterical palpitation come under this head. Hysteria is frequently, but by no means invariably, associated with anæmia. It occurs in the plethoric. Functional disorder of the heart is one of the commonest of the varied phenomena included under the name of hysteria. Disturbed action of the heart is often a prominent feature of the hysterical condition. Various morbid agencies induce a state of nervous derangement, of which functional disorder of the heart is a distressing manifestation. Venereal excesses and the solitary vice are frequent causes. In the endeavor to trace this and other effects of derangement of the nervous system to their source, the practitioner should not omit inquiries as to sexual indulgence in the married, for there are persons who appear to think that any amount of

legitimate indulgence is innocent, and when questioned, will confess to having practised one or more acts of coition daily for a series of years. The excessive use of tobacco is another cause of nervous derangement giving rise to functional disorder of the heart. This is a frequent cause. Many persons are led by their experience to observe that after an unusual indulgence in this luxury, they are apt to suffer from palpitation, and the disorder is sometimes removed by simply discontinuing this indulgence. Strong tea in some persons occasions severe paroxysms of palpitation. Dr. Stokes has cited several striking illustrations.[1] Strong coffee induces this effect in certain conditions of the system, or in consequence of a peculiarity of constitution. Excessive mental exercise and protracted vigilance belong in this category; and, more than all, long continued anxiety or distress of mind. In a pretty large proportion of the cases of functional disorder of the heart, it is traceable to nervous derangement induced by mental causes. Persons are especially prone to this disorder who are so constituted that, whatever may be the circumstances surrounding them, they are constantly anxious and worried. In persons not thus unhappily constituted, the disorder may originate in the severe afflictions, calamities, and disappointments to which human life is exposed. Whatever may be the causes inducing that derangement of the nervous system which leads to disturbed action of the heart, mental depression is generally a prominent symptom. The conviction of the existence of organic disease is often with great difficuly removed. The patient sometimes persists in this conviction in spite of the strongest assurances of the physician. His attention is occupied much of the time in watching the action of the heart. He acquires the habit of feeling the pulse or the beating in the præcordial region. He lives in daily apprehension of sudden death. This truly pitiable condition tends, in no small degree, to aggravate the nervous derangement, and thus reacts on the cardiac disorder. Every one who has been brought much into contact with students of medicine, must have been led to remark the frequency with which they imagine themselves to be affected with disease of the heart. The study of the diseases of this organ tends to direct attention to the subject and excite their fears, if, from any cause or combination of causes, functional disorder is produced; and the dread

[1] Dr. Stokes devotes a section to "Disturbance of the heart caused by the use of tea." *Vide* On Diseases of the Heart and Aorta, Am. ed., p. 533.

of these diseases seems, in some instances, to be alone sufficient to induce disturbed action of the organ. A fixed belief that the heart is diseased is one of the commonest of the delusions incident to melancholia and hypochondriasis.

Disorder of the heart often accompanies dyspeptic ailments. It appears to be produced through the sympathetic relations existing between the heart and stomach. Paroxysms of palpitation are frequently referable to a fit of indigestion. The latter may be the immediate determining cause in cases in which the disorder involves other causative conditions than dyspepsia. Dyspeptic ailments, in fact, in a large proportion of cases, proceed from derangement of the nervous system, induced especially by mental distress or anxiety; and it is not easy to say, under these circumstances, to what extent the cardiac disorder is dependent on a morbid condition of the stomach. Dyspeptics who suffer from disturbed action of the heart are apt to insist perseveringly on the existence of organic disease, and to cherish the most gloomy forebodings. They fall into the baneful habit of watching the action of the heart by placing the hand over the præcordia or on the pulse, and listening, at night, to the cardiac sounds. Under these circumstances they find evidence of disorder, because the anxious expectation of finding it is often sufficient to produce it.

The accumulation of gas in the stomach, when other dyspeptic symptoms are not present, seems often to produce or increase cardiac disorder. This may be owing to mechanical pressure upon the heart. Patients suffering under paroxysms of palpitation frequently make voluntary efforts to expel wind from the stomach by belching, and express relief when they succeed in these efforts. Carminative remedies, in many instances, are useful in this way. Gastric distension, in many cases of hysteria, aggravates the symptoms referable to the heart.

The gouty diathesis involves a liability to functional disorder of the heart. Paroxysms are apt to precede other manifestations of this diathesis, occurring before any affection of the joints takes place, and perhaps ceasing to recur after the latter becomes established. Palpitation is sometimes a premonition of an approaching fit of gout. It may occur also in the intervals between the gouty affections. The disorder, in persons subject to gout, may be due to other morbid conditions—for example, plethora; but it is reasonable to conclude, from the relations often observed to exist between the disturbed action of the heart and the arthritic attacks, that the

former arises from the accumulation in the blood of the poisonous agent—supposed to be lithic acid—which gives rise to the latter. This view of the pathology has an obvious practical bearing on the management.

Other pathological relations of functional disorder of the heart have been noticed by clinical observers. It occurs during convalescence from fevers. Persons affected with deformities of the chest seem to be more liable to it. Corrigan and Forget have noticed its frequent occurrence in young persons when growth is unusually rapid. It is, apparently, sometimes induced by excessive muscular exercise. An abnormally small size of the heart has been supposed to contribute to its production. It is probable, however, that, when developed under these and other circumstances which might be added, the immediate causative conditions are included under the several classes which have been noticed, consisting of abnormal changes pertaining to the blood, or derangement of the nervous system induced by various morbid agencies, or disturbing influences, transmitted, by sympathy, from other organs.

Symptoms of Functional Disorder of the Heart.

The symptomatic phenomena in cases of functional disorder differ materially according to the various pathological relations in which it is presented in practice. Associated with plethora, it is accompanied by symptoms denoting vascular fulness, such as a strong pulse, a flushed face, cephalalgia from determination of blood to the brain, frequently obesity, etc. In connection with anæmia, the attendant phenomena indicate feebleness of the circulation; the lips are pallid, the pulse small and quick, the extremities cold, etc. Dependent on certain derangements of the nervous system, it forms, in some cases, one of the multifarious elements of hysteria; in other cases, hypochondriasis, melancholia, and other symptoms referable to this system, are prominent. As incidental to dyspepsia, it is conjoined with notable disorder of the digestive functions. Occurring in persons subject to gout, it is either combined or alternates with the varied ailments incident to this diathesis. These diversified phenomena are not properly symptoms of the cardiac disorder, but pertain to the different morbid conditions which give rise to it. And, in fact, as already stated, functional disorder of the heart is merely a symptom of these morbid condi-

tions, and not entitled strictly to be considered as a cardiac affection. There are certain points, however, pertaining to symptomatology, which are of importance in discriminating functional disorder from organic disease. These it will be most convenient to notice in connection with the subject of diagnosis.

Physical Signs of Functional Disorder of the Heart.

Physical exploration will be seen more fully under the head of diagnosis to be of immense value in cases of functional disorder of the heart, as showing the absence of the signs of inflammatory and organic affections. The information which it affords is not less positive than if there were certain signs characteristic of functional disorder. The results of physical exploration are, in fact, to be considered under a twofold aspect, viz., *first*, as to the absence of abnormal phenomena which denote structural changes; and, *second*, as to the presence of the normal phenomena denoting soundness of the organ. In the latter point of view the evidence is positive, in the former it is negative. It is of use, practically, to keep this distinction in mind. In exploring the chest, the practitioner has always two objects in view. One object is to ascertain whether certain well-established signs of disease are either present or wanting; another object is to satisfy himself as to the presence of the healthy signs. To illustrate this distinction, if solidification of lung be suspected, auscultation is practised in order to discover the respiratory sign of solidification, viz., the bronchial respiration. Now, every practical auscultator knows that the lung may be solidified, and yet this sign of solidification be wanting. The evidence against solidification, therefore, is not complete when it is found that this sign is absent. But let it be ascertained that, in place of a bronchial respiration, the normal respiratory murmur continues, here is proof positive of the non-existence of solidification. This principle will be found to apply to the employment of physical exploration with a view to determine whether certain symptoms referable to the heart proceed from organic disease or merely functional disorder.

Of the several methods of exploration, percussion, palpation, and auscultation furnish important information in cases of functional disorder.

By percussion it is ascertained that the heart is not enlarged. Functional disorder, it is true, may coexist with cardiac enlarge-

ment, the combination being due merely to coincidence. It does not follow because the heart is found to be enlarged and other lesions of structure are present, that functional disorder, irrespective of the organic disease, does not exist. But absence of enlargement is presumptive evidence that the disorder is purely functional; for clinical experience teaches that in cases of disturbed action of the heart arising from organic disease, the latter generally has induced enlargement of the organ. Percussion, therefore, is of great utility in the discrimination of functional disorder from affections involving lesions of structure.

The abnormal force of the heart's action is ascertained by palpation. The impulsion in severe paroxysms of palpitation is often violent; the whole præcordia is agitated; the organ seems to strike a forcible blow against the thoracic walls. The irregularity of the movements of the organ is also appreciated by the hand. These are merely signs of increased and disturbed *action* due to morbid excitement of the heart. They do not indicate the augmented *power* of the organ, which characterizes hypertrophy. The impulse in hypertrophy denotes strength rather than force; it is not quick and violent, but sluggish and strong; it does not give the sensation of a shock or blow, but it causes a gradual and powerful heaving of the præcordia. The characters obtained by palpation, which distinguish functional excitement of the heart from enlargement by hypertrophy, are sufficiently well-marked, and have been mentioned already in treating of the latter.[1] The discrimination, however, does not rest on this distinction, for the fact that enlargement exists, in cases of hypertrophy, is determined by the coexistence of other signs. But it is to be borne in mind that functional disorder dependent on some of the morbid conditions which give rise to it independently of organic disease, may be associated with hypertrophy, and, under these circumstances, the excited action due to the former, and the increased power due to the latter, are combined.

Palpation shows, in cases of functional disorder exclusive of organic disease, that the point of apex-beat is in its normal situation; not elevated as in pericarditis with effusion, nor lowered and carried to the left as in cases of enlargement of the left ventricle.

Purring tremor, or thrill, is said to be sometimes perceived at the base of the heart in cases of purely functional disorder. This must be extremely rare. Well-marked thrill is to be considered as a sign of hypertrophy of the left ventricle combined with valvular

[1] *Vide* Chapter I. p. 51.

lesions. Auscultation furnishes important information, *first*, negatively, by showing the absence of adventitious sounds indicative of valvular lesions, and, *second*, positively, by showing that the natural sounds preserve their essential characters and normal relations to each other.

As regards adventitious sounds, the question arises, may not an endocardial murmur be produced by functional disorder alone? It is supposed that a mitral systolic murmur sometimes occurs in paroxysms of palpitation, in consequence of spasmodic action of the papillary muscles connected with the mitral valve, interfering with the action of the latter sufficiently to permit a certain amount of regurgitation irrespective of any valvular lesions. Without denying the possibility of this occurrence, it must be extremely rare, and a murmur thus produced is necessarily either intermittent or of a transient duration. A murmur referable to the mitral orifice, in the vast majority of instances, proceeds from physical changes, although these may be trivial as regards any immediate effects; and if the murmur be persistent, it certainly denotes lesions, either innocuous or otherwise. At the arterial orifices, viz., the pulmonic and aortic, a murmur is often present in connection with functional disorder of the heart, when there are no valvular lesions in these situations. This murmur is therefore inorganic, and in the great majority of cases it is dependent on the condition of the blood. The very frequent association of functional disorder with anæmia, accounts for the frequency of the murmur. May not the murmur in some instances be dynamic, *i. e.*, due to the excited action of the heart, without involving an abnormal condition of the blood? The affirmative is not improbable, but it is difficult to answer this inquiry positively, and practically it is not very important to do so. The question to be settled, clinically, in individual cases is, whether a murmur referable to the aortic or pulmonic orifice, coexisting with disturbed action of the heart, be organic or inorganic. The points involved in the discrimination of organic and inorganic murmurs have been considered in a preceding chapter.[1] These points may be here briefly recapitulated. An inorganic murmur is always systolic, and very rarely, if ever, rough in quality. Assuming that it is produced at the arterial orifices, and therefore seated at the base of the heart, it may be referred to the aorta or pulmonic artery, either or both; if the latter, this fact renders its inorganic character almost certain, provided congenital valvular

[1] Chapter IV. p. 202.

lesions are excluded. An arterial murmur is heard over the carotids, and perhaps over other large arteries which are accessible.[1] Venous hum in the veins of the neck, especially on the right side, coexists in the great majority of instances. The murmur is usually feeble, and variable in intensity; it is often intermittent. The palpable evidences of anæmia are usually present, and it occurs much oftener in females than in males. An organic murmur, on the other hand, may be diastolic, or systolic and diastolic murmurs may be combined. It is often rough or musical. It is referable to the aortic orifice, if not to the mitral, unless it be dependent on congenital valvular lesions. If not propagated into the carotids, murmur in this, as well as in other arterial trunks, may be wanting. Venous hum may not coexist. The murmur is persistent and less fluctuating as regards intensity. Anæmia is often not apparent.

Attention to these differential points will generally enable the practitioner to discriminate correctly between an organic and inorganic murmur; but this discrimination, practically, with reference to the question, whether disturbed action of the heart be due purely to functional disorder, or not, is of less importance than might at first be supposed. The disturbance is probably dependent on functional disorder, whether an existing murmur be organic or inorganic, if the heart be not enlarged. It may be stated, as a rule, that valvular lesions do not give rise to notable disturbance of the heart's action prior to more or less enlargement. Hence, cardiac disorder in a marked degree, when valvular lesions exist, is attributable to abnormal conditions which are independent of the latter. The fact already repeated more than once is not to be lost sight of, that the causes of functional disorder may be superadded to organic disease; in other words, that structural lesions do not render the heart exempt from the liability to become functionally disordered in consequence of the same causes which occasion disturbance of its action when it is structurally sound.

The heart-sounds in cases of functional disorder, preserve essentially their normal characters. They are, however, intensified in proportion to the increased force of the heart's action. Their intensity is often such that they are perceived by the patient with great distinctness, especially at night. The beating of the heart is sometimes distinguished by others at some distance from the

[1] It is to be borne in mind that an arterial murmur may be produced simply by pressure over the artery with the stethoscope.

chest. The valvular element of the first sound is in some cases unusually developed, owing to the abnormal force and quickness of the systolic contractions, and it may predominate over the element of impulsion, rendering this sound short and valvular in quality like the second sound. The predominance of the valvular element of the first sound may thus occur in opposite conditions as respects the muscular action of the heart, viz., when it is enfeebled, and when it is excited. The first sound, more than the second, is affected in its intensity, by the vital condition of the heart. It is relatively weakened, and may be suppressed when the muscular power of the organ is greatly reduced. On the other hand, it becomes the accentuated sound at the base, and at points removed from the præcordial regions, when the muscular action is increased by morbid excitement. The integrity of the heart-sounds; the normal relative intensity of the aortic and pulmonic second sound, and of the mitral and tricuspid elements of the first sound, constitute important evidence, in cases of disturbed action of the heart, that the latter is due to simply functional disorder.

The apex-beat, or systolic sound of the heart, is sometimes accompanied by a ringing intonation called by Laennec *cliquement metallique*, or metallic tinnitus. This is occasionally observed to some extent, in health, especially in young persons, even when the heart is tranquil. It is, however, in general, a sign of excited action of the organ. It may be imitated by making light percussion on the back of the hand, the palmar surface being applied over the ear. Hope explains the production of this metallic ringing sound by supposing that "the heart in gliding forwards and upwards during its systole strikes with its apex against the *inferior margin* of the fifth rib, and thus creates an accidental sound, attended by *cliquetis* when the blow is smart." He adds: "It may be prevented at pleasure by pressing the edge of the stethoscope or anything else into the intercostal space by which that space is put, internally, on the same plane or the rib over which the heart then glides without catching." If this be the correct explanation, inasmuch as the heart does not move upwards and forwards during its systole, the sound must be due to the apex impinging against the upper margin of the sixth, rather than the lower margin of the fifth rib, that is, assuming the point of apex-beat to be in the fifth intercostal space, as it is in the majority of persons. Whatever may be the explanation, clinical observation shows that the sign occurs when the action of the heart is abnormally quick and forcible, and that it is produced by the

movements of the apex against the thoracic walls, can hardly be doubted. It may occur in cases of hypertrophy, but it is more apt to be developed in connection with merely functional disorder, and it is, therefore, to some extent, significant of the latter. It was stated by Hope, that he never found it to occur in any but the meagre. It occurs certainly very seldom in persons whose chests are thickly covered with muscle or fat. Tympanitic distension of the stomach contributes to the intensity and clearness of the sound, and it may occur only under this condition. In a case observed by Dr. Walshe the sound was so loud as to be a source of alarm to the patient. Dr. Stokes remarks, and justly, that it is more common in cases in which the heart acts with great force combined with regularity of action, than in the irregularly acting hearts. As a physical sign, tinnitus is not of much practical value, since it may occur when the heart is excited, and, under circumstances, when it is tranquil, in health, and since it occurs in cases of enlargement as well as of merely functional disorder, although more frequently in the latter. It is perhaps important to warn the inexperienced auscultator not to attach to it a degree of significance as a morbid sound to which it is not entitled.

Diagnosis of Functional Disorder of the Heart.

The diagnosis of functional disorder of the heart involves in all cases the question whether organic disease be or be not present. The symptomatic phenomena referable to the heart are sufficiently explicit as to their source. The patient, as well as the physician, is able at once to determine their cardiac origin. But whether these phenomena proceed merely from disturbed action, or are due to a structural affection, is not so easily determined. The question is one of great practical importance. If there be only functional disorder, the physician is warranted in giving positive assurances of the absence of danger, and in holding out confident expectations of recovery. If organic disease be present, such assurances and expectations are not admissible. An intelligent patient is sufficiently aware of the difference between organic disease and functional disorder to appreciate its great importance; and he anxiously appeals to the physician for positive information with respect to this point. The ability to say positively that organic disease does not exist, often enables the physician to exert a moral influence of

no mean value upon the continuance of the malady, as well as in rendering it more supportable. Errors in diagnosis are quite common. Instances have repeatedly come under my observation in which patients suffering only from disturbed action of the heart, having been told that they were affected with organic disease, have lived for months or years under a sense of danger of sudden death, a condition of mind highly conducive to the perpetuation of the disorder. On the other hand, it is not uncommon for the symptoms connected with structural lesions to be imputed to merely functional disorder. The latter error, although less unfortunate as regards its consequences than the former, sometimes leads to evil results. If the physician be not confident in his ability to decide as to the existence or non-existence of organic disease, but is sufficiently prudent not to commit himself to any conclusion, he loses the advantage which he might avail himself of, assuming the affection to be merely functional, and the patient naturally construes his reserve or indecision into an unfavorable opinion. In short, there are few problems in clinical medicine more important than that which calls for a decision as to the existence of a purely functional disorder of the heart, or an organic affection; and this problem cannot fail to present itself very frequently in medical practice. Cases of organic disease of the heart are not infrequent, and cases of merely functional disorder are exceedingly common. The importance of the diagnosis must be felt almost daily by the reflecting and conscientious practitioner.

But the diagnosis involves more than the question whether disease be or be not present. Functional disorder may be superadded to organic disease. The latter may exist, but not to an extent to occasion immediate inconvenience or danger, the symptomatic phenomena being due to disturbed action arising from morbid conditions, independently of the structural lesions which happen to coexist. The fact that functional disorder and organic disease may be associated, and the former not dependent on the latter, is not to be lost sight of. Hence, it is not enough to decide that organic disease is present; the question then arises, Is this organic disease the source of all the symptomatic phenomena referable to the heart, or are they not due, in a greater or less degree, to functional disorder dependent on morbid conditions which have no connection with the cardiac lesions? This is a question of great importance, which is to be considered in the cases of disturbed

action of the heart, in which the evidence of organic disease is found to coexist.

The objects in diagnosis, then, are, *first*, to determine whether organic disease be or be not present; and, *second*, if organic disease be present, to determine whether superadded functional disorder be not the source of more or less of the symptomatic phenomena referable to the heart.

The symptoms referable to the heart, separately or combined, cannot afford positive evidence in any case that cardiac disorder is purely functional. Yet there are several points pertaining to the symptomatology, exclusive of physical signs, which are consistent with the supposition of the existence of functional disorder rather than of organic disease. These points are to be considered with reference to the diagnosis.

The mental condition is of some importance in a diagnostic point of view. Functional disorder generally occasions, in a marked degree, anxiety and apprehension. The patient is often much agitated by the idea of an examination, and awaits the result with fear and trembling. It is not infrequently difficult to convince him that he has not an organic affection, and he sometimes solicits repeated examinations lest something may have been overlooked. On the contrary, patients affected with organic disease are often, in a marked degree, apathetic on the subject. They are inclined to think that their ailments proceed from some other organ than the heart, for example, the liver or the stomach. They generally bear being told that the heart is unsound, without emotion, and frequently with apparent indifference. The contrast in the state of the mind with reference to the question as to the existence or non-existence of organic disease, is very striking.

The symptoms due to disordered action of the heart from organic disease, viz., palpitation, irregularity, intermittency, etc., occasion, as a rule, far less inconvenience than when similar symptoms arise from merely functional disorder. It is surprising, in some cases, to what extent the action of the heart is disturbed in connection with structural lesions, without the patient apparently being conscious of it. Power of impulse sufficient to raise the præcordia, and jar the whole body, is sometimes unnoticed. Irregular and intermittent action does not excite fear of sudden death. It is otherwise with cases of functional disorder. Palpitation, in these cases, causes great distress; and rhythmical disturbance produces fear that the action of the heart may be suspended, and a feeling of impending

dissolution. The positive suffering from symptoms referable to the heart, and the mental condition, furnish strong presumptive evidence of the existence of merely functional disorder.

The paroxysmal character of functional disorder, and the complete exemption, at certain periods, from cardiac disturbance, are important diagnostic points. Structural lesions, being permanent, if they are sufficient to occasion much obstruction or regurgitation, or both, induce, at length, certain symptoms which are constant, such as feebleness, irregularity and intermittency of the pulse, dyspnœa on exercise, etc. Functional disorder, on the other hand, occurs, generally, in well-marked paroxysms, and after these have ceased, the action of the heart may be natural, and there are no symptoms referable to this organ habitually present. A patient who is able, at any time, to take active exercise without undue excitement of the heart, or dyspnœa, may be presumed to be free from organic disease. But it is not safe to rely on the statements of patients with respect to this point, for persons affected with organic disease are often unconscious of these effects of exercise, when they are sufficiently apparent to others. Persons liable to functional disorder often are not only able to engage, without discomfort, in pursuits requiring great muscular exertion, but they are less likely, under these circumstances, to suffer from cardiac disturbance. The obvious benefit of active exercise thus becomes, in some measure, diagnostic. But the want of ability to take active exercise is by no means proof that organic disease exists, for in some cases of functional disorder associated with anæmia, slight exertion may induce palpitation, dyspnœa, etc.

Certain symptomatic events belong especially to the clinical history of organic affections, and not to that of functional disorder. Thus, general dropsy very rarely occurs in connection with the latter. This is true of lividity, hæmoptysis, paralysis from embolia, etc. These events point to the existence of organic disease, but their absence does not prove that merely functional disorder exists, for they by no means accompany invariably structural lesions.

It has been seen that functional disorder has certain pathological relations. The presence of the morbid conditions in connection with which it is apt to occur, is to be taken into account in the diagnosis. Thus, cardiac disturbance is presumably functional, if it be connected with plethora, anæmia, derangement of the nervous system from excessive venery, mental anxiety, the use of tobacco,

etc., dyspepsia or gout. On the other hand, organic disease, in a large proportion of cases, originates in acute rheumatism. Hence, if a patient have never had the latter affection, the fact increases the chances that the cardiac disorder is merely functional.

The age of the patient is to be considered. Functional disorder occurs especially in the young, or between the age of puberty and middle life. Organic disease is oftener presented during or after the middle period of life. Functional disorder is oftener met with in females than in males; the reverse being true of organic disease. Organic disease occurs more frequently among the laboring classes of society, especially those exposed to the vicissitudes of the weather; functional disorder is more common among the sedentary and luxurious.

Disturbed action from functional disorder is apt to occur especially at night, probably because the mind of the patient being abstracted from outward objects, the attention is more likely at this time to be directed to the heart, or his thoughts are more concentrated on himself. Persons with organic disease experience more inconvenience during the day-time, when they are exposed to causes which excite the circulation, such as exercise. Disturbance of the heart's action beyond that which is habitual, in persons affected with organic disease, is generally proportionate to obvious exciting causes. On the other hand, the action of the heart in cases of functional disorder is often out of proportion to appreciable causes; a sudden start, for example, sometimes occasions violent palpitation. Severe paroxysms of functional disorder often are not attributable to any apparent exciting cause.

The foregoing points are to be considered in the discrimination of functional disorder from organic disease; but, singly or collectively, they are never sufficiently diagnostic to warrant a decision that organic disease is not present. A positive diagnosis demands the information to be derived from physical exploration. The latter affords the readiest as well as the only sure way of coming to a decision. The employment of physical exploration in cases of merely functional disorder is one of the most beautiful (if this expression may be allowed), as well as useful, of the practical applications of this method of examination. A few moments often suffice to decide that the heart is free from structural lesions; and, reasoning by way of exclusion, that the symptoms referable to the heart are consequently due to functional disorder only.

In excluding organic disease, the absence of physical signs refer-

able to structural lesions is to be ascertained. Is the heart enlarged? This is to be determined by defining the boundaries of the superficial and deep cardiac regions by means which have been fully considered in Chapter I., and by ascertaining that the point of apex-beat is within the range of healthy variations. Does auscultation fail in detecting adventitious sounds or murmurs? This is almost, if not quite, enough to warrant the conclusion that valvular lesions do not exist. Are murmurs discovered? Then it is to be determined whether they are organic or inorganic. The differential points involved in this discrimination have been mentioned in another division of this chapter. The exclusion of organic disease is rendered more positive by ascertaining, not only the absence of the physical signs denoting structural lesions, but the normal character and relations, in all essential particulars, of the heart-sounds. These are to be observed in different situations, the auscultator interrogating, successively, the aortic, pulmonic, mitral, and tricuspid valves, in the manner already described.

But let it be assumed that organic disease is not excluded; in other words, that the signs of structural lesions are present. It is to be determined whether functional disorder be not superadded. This is to be done by comparing the amount of organic disease with the degree of disturbed action. If the latter be disproportionate to the former, it is probably due, in a great measure, to functional disorder dependent on other morbid conditions than the lesions of structure. The amount of organic disease and the effects which are fairly attributable to them may be ascertained, approximatively, by means of the physical signs. Is the heart but little, if at all, enlarged, and do the heart-sounds preserve their normal characters and relations to an extent showing that the lesions cannot involve, to much extent, obstructive or regurgitant effects; disturbed action, if excessive or considerable, is probably due mainly to superadded functional disorder. It is important, in this connection, to take into view the presence or absence of the morbid conditions which are likely to give rise to functional disorder, viz., plethora, anæmia, etc. The presence of these conditions adds much to the probability of the symptomatic phenomena referable to the heart being due to functional disorder. It is a common error to attribute all these phenomena to the lesions of structure, whenever the existence of the latter is determined—an error often unfortunate as regards the prognosis and treatment. The lesions may be innocuous, and the cardiac symptoms dependent altogether on

coexisting functional disorder. It is to be borne in mind that structural lesions, as a rule, do not give rise to disturbance of the heart's action sufficiently to occasion much, if any, inconvenience, prior to enlargement of the organ; and not infrequently the organ becomes considerably enlarged before the attention of the patient is awakened to any symptoms denoting an abnormal condition of the heart.

The discrimination of cases of fatty degeneration of the heart from those of purely functional disorder is sometimes attended with difficulty. The difficulty arises from the fact that this form of organic disease does not present any positive physical signs. It is, therefore, not so easily excluded as are valvular affections and uncomplicated enlargement of the heart. In most instances, however, enlargement coexists with fatty degeneration, and not infrequently lesions of the valves are also conjoined. Exclusive of these complications, the symptomatic phenomena referable to the heart in cases of fatty degeneration are analogous to those which denote functional disorder. This structural change occurs at a period of life when persons are not so much exposed to merely functional disorder as at an earlier age. The palpitation connected with it has not that violence which frequently characterizes disturbed action when the muscular structure is sound. The paroxysmal character of merely functional disorder is less marked. Feebleness of action, and perhaps irregularity, are permanent symptoms. These circumstances, taken in connection with the various events which have been noticed under the head of the pathological relations and effects of fatty degeneration, in Chapter II., will generally enable the physician to determine whether this affection be or be not present. But it is to be borne in mind that the morbid conditions giving rise to functional disorder may be associated with fatty degeneration, as well as with other varieties of cardiac lesion.

Prognosis in Cases of Functional Disorder of the Heart.

The prognosis in cases of functional disorder of the heart is always favorable. Although the irregularity and violence of the disturbed action are sometimes such as apparently to involve immediate danger, it is doubtful whether a paroxysm ever proved fatal; nor do any unpleasant results follow, except a certain amount of exhaustion and nervous excitement. Recovery from the morbid

irritability of the organ may be expected, but it often tends to continue for a considerable length of time. Of this the physician should be aware, and it is well to forewarn the patient that the duration of his malady may be tedious. After being assured, however, that he is not affected with an organic disease, and finding, by experience, that paroxysms occur and pass off without accident or injury, he endures their recurrence with greater patience than at first, and, at length, if they are not severe, he comes to regard them with comparative indifference.

It was formerly supposed that functional disorder, if protracted, eventuates in organic disease. This doctrine has been disproved by clinical experience. There is no ground for the belief that changes of structure ever originate in disturbed action of the heart, however persisting. I have known persons who have suffered from attacks of palpitation, frequently repeated for many years, without enlargement, or other lesions becoming developed. In cases in which inordinate action has continued steadily for several successive months, the soundness of the organ has remained unimpaired, and complete recovery has taken place. It is pleasant, as well as useful, to be able to assure patients affected with functional disorder that they are not rendered thereby liable to organic disease.

Treatment of Functional Disorder of the Heart.

Therapeutical indications in cases of functional disorder of the heart, relate to two objects, viz: *First.* Relief of disturbed action when present. This object embraces palliative measures only. *Second.* Removal of the morbid irritability of the organ. This object embraces curative measures, in other words, those by which it is expected recovery will be effected.

Curative indications are derived chiefly from the pathological relations and causes of functional disorder. The abnormal conditions with which morbid irritability of the heart is connected, being different in different cases, the treatment cannot, of course, be uniform. The measures of therapeutics, in fact, differ, in individual cases, not less than the conditions on which cardiac disorder is dependent.

When associated with plethora, depletory measures are indicated. Bloodletting, locally or generally, is judicious in some cases. It should, however, be employed with circumspection. Resorted to

when not indicated, or carried too far, it tends to aggravate the cardiac disorder. This is shown by the effect of hemorrhages, and of the injudicious employment of bloodletting, formerly more than now, in various affections. The "reaction from loss of blood," as illustrated by the researches of Marshall Hall, and others, expresses phenomena which are mainly due to abnormal irritability of the heart. The existence of plethora is to be clearly ascertained before resorting to bloodletting, and it is to be borne in mind that the cases in which this condition of the blood exists, are comparatively few in number. In most instances, if the existence of plethora be sufficiently evident, an adequate amount of depletion may be received by saline laxatives and a reduced diet. The latter methods of depletion must not be pushed too far, or continued too long. The limit is the restoration of a normal condition of the blood. If the proportion of red globules be reduced below that of health, there is risk of the cardiac disorder being increased rather than diminished. When the proper limit is reached, habits of active exercise are to be conjoined with a nutritious, but not over-generous diet. Animal food should be taken sparingly, and alcoholic stimulants avoided. These are the measures indicated by the coexistence of plethora.

Associated with anæmia, which is vastly more frequent, the measures indicated are the reverse of those appropriate when plethora exists. The treatment now should be directed with a view to increase the proportionate quantity of the red globules of the blood. For this end, tonic remedies, and especially preparations of iron, are to be employed. The diet should be highly nutritious, and consist of a good proportion of animal food. Alcoholic stimulants, in the form of spirits, wine, beer, or porter, are generally useful. Moderate exercise in the open air is to be enjoined. The causes which have induced, and which may perpetuate the anæmic condition, are to be ascertained, and, if possible, removed. This will embrace the appropriate treatment of various local affections which in females are often seated in the genito-urinary system, such as leucorrhœa, menorrhagia, etc.; weaning in certain cases, avoidance of pregnancy, and, in short, proper attention to all the various circumstances which, in different cases, may be involved in the production and continuance of the anæmia. Bloodletting and other measures of depletion, in cases belonging to this class, are positively pernicious, and may prove so in a marked degree. The discrimination of these cases from those in which the cardiac disorder is connected with plethora, is highly

important with reference to appropriate treatment. The fact that, in the great majority of instances, functional disorder of the heart is more or less dependent on anæmia, is not to be lost sight of.

Derangements of the nervous system arising from different causes, can only be treated successfully when the latter are ascertained and removed. Until these ends are attained, the cardiac disorder will be likely to continue. The general indication, in the cases coming under this head, is to place the patient without the influence of certain morbid agencies. The most prominent of these are, sexual excesses, the abuse of tobacco, tea, or coffee, excessive mental exertion, vigilance, and mental anxiety from a variety of causes, real or imaginary. Curative measures consist in removing these causes, so far as they are controllable, together with the employment of remedies, and a regimen calculated to restore the healthy condition of the nervous system. Change of scene, the excitement of travel, and recreation, are often highly useful, and may be sufficient to effect recovery, when the disorder depends mainly on causes pertaining to the mind, as is not unusual. The importance of inquiry with respect to sexual excesses, is to be borne in mind. I have met repeatedly with cases in which cardiac disorder was traceable to this source. As already remarked, it appears to be an impression with some persons that indulgence cannot be excessive except when it is meretricious; hence, it is not enough to know that a patient is married. Under the head of sexual excesses, self-pollution is included. Of the difficulty often in obtaining information concerning this matter, especially with regard to females, it is unnecessary to speak. This may account for the obstinacy with which functional disorder of the heart persists in certain cases.

The coexistence of dyspeptic ailments calls for a proper regulation of diet and regimen, together with remedies to relieve gastric derangements and improve digestion. So far as the cardiac disorder depends on functional disturbance of the stomach, the treatment resolves itself into that due to the latter, of which the former is but a symptom. To consider the treatment of dyspepsia, would be here out of place. It may, however, be remarked that the ailments comprehended by this term generally involve morbid conditions seated elsewhere than in the affected organ, and often depend on mental causes. The treatment, therefore, must have reference to these ulterior conditions. In the dietetic management, it may be added, the object is not to reduce the diet to an extent corresponding to the weakened digestive power, but to invigorate and

strengthen the latter, so that ordinary wholesome articles of food may be taken without inconvenience. The measures for this end are, tonics, stimulants, exercise in the open air, mental recreation, and persistency in a nutritious and varied diet, in spite of occasional symptoms denoting difficult or imperfect digestion. It is mistaken policy to watch the effects of taking particular articles of food, and eliminate, successively, from the diet, those which are found to occasion inconvenience. Various accidental causes may disturb digestion, and a meal which on one day may be followed by distress, on the next day may be taken without trouble. The practical rule for the dyspeptic, as regards diet, is to eat the ordinary, wholesome, well-cooked varieties of food in sufficient quantity to meet the wants of the system, trusting to remedies and regimen to render the digestive organs adequate to the performance of their duty. These remarks are, of course, only applicable to cases of merely functional disturbance of the stomach.

Functional disorder of the heart occurring in persons affected or threatened with gout, claims the remedies which are indicated by the gouty diathesis. These are medicines supposed to act by elimination, of which the most efficient is colchicum, and alkalies given with a view to neutralize the excess of lithic acid in the blood. Of the latter, potash is considered as preferable to soda, in consequence of the solubility of the salt formed by the union of lithic acid with the former. Alkaline remedies and colchicum may be combined. The iodide of potassium has been found useful. The mineral saline waters are especially suited to this class of cases.

The several morbid conditions which give rise to functional disorder of the heart, may be more or less combined in certain cases. Under these circumstances, the treatment must have reference, of course, to the different conditions existing in combination. The gouty diathesis, for example, may be conjoined with plethora, or dyspepsia; dyspepsia and anæmia are often united, etc. It is not enough to have ascertained the existence of one of the conditions upon which cardiac disorder may be dependent; the inquiry is to be extended so as to embrace others which may coexist.

One of the most important of the means of promoting recovery, is applicable to cases occurring in each and all of the different pathological connections. This is the moral influence of a positive assurance that the heart is free from organic disease. The anxiety and apprehension incident generally to disturbance of the heart's action, tend powerfully to perpetuate and aggravate the disorder.

If the physician be sufficiently confident in his diagnostic ability to assure the patient confidently that the affection is purely functional or inorganic, and the patient have sufficient confidence in the knowledge and judgment of the physician to believe the assurance, this will often go very far toward promoting a cure. The effect, in many instances, is truly remarkable. Hence, a great practical advantage is to be derived from a sure diagnosis. An opposite effect is equally marked when the patient is told that he has organic disease. I have met with instances in which several years were embittered by a false diagnosis and its imprudent communication. This, with the measures generally advised in conjunction, suffices to perpetuate the disorder indefinitely. If the physician be competent to employ physical exploration, and to satisfy himself thereby that organic disease does not exist, he should take pains to remove the groundless fears of the patient, at the same time forewarning him that the disorder is liable, when it once occurs, to continue for a greater or less period. If the patient can be made to believe that there is no danger, the malady is rendered supportable until recovery is effected. This is a point in the treatment of functional disorder of the heart, second to none other. In all cases the patient should be advised not to watch the action of the heart by feeling the pulse or the apex-beats, or listening to the sounds at night. His attention should be diverted from the disordered organ as much as possible. The benefit of agreeable occupation is, in part explicable by its effect in this way.

Functional disorder, not dependent on, but coexisting with organic disease, claims essentially, the same measures as when disconnected from the latter. With reference to treatment, it is highly important to determine that functional disorder from some one or more of the morbid conditions which give rise to it, is superadded to organic disease. I have met with instances repeatedly in which, in consequence of this combination, patients appeared, at first view, to be in an advanced stage of organic disease, but who recovered, by judicious management, apparently, perfect health. The practitioner cannot be too often cautioned not to attribute all the symptomatic phenomena referable to the heart to structural lesions, when the latter are found to exist. The practical rule may be here repeated, to regard these phenomena as probably due to functional disorder whenever the heart is but little, if at all, enlarged. The association of anæmia with a certain amount of organic disease is quite common, when if the blood be restored

to its normal condition, the cardiac lesions are found to be, for a time at least, innocuous. But if the lesions involve more or less obstruction or regurgitation, and enlargement of the heart have already taken place, marked improvement, as regards the cardiac and other symptoms, may be expected to follow the removal of the conditions which give rise to the associated functional disorder.

The foregoing remarks have had reference to measures which are distinguished as curative. Palliative measures are now to be considered. These are to be adapted to different circumstances pertaining to the disturbed action of the heart. To tranquillize the excited organ and restore regularity of action, are the ends for which palliative measures are pursued. But the heart is subject, as has been seen, to various forms of disorder, and the condition of the organ, as manifested by the symptoms, is not precisely the same in all the several varieties. In the mildest form of disorder, in which only an occasional, momentary disturbance is felt, there is neither necessity nor time for palliation. Curative measures are alone required. Persisting, inordinate action, continuing perhaps for weeks or even months, calls for remedies calculated to allay this state of excitement. The special cardiac sedative, as it has been called, digitalis, is often useful for this purpose. Hydrocyanic acid, or the laurel water, hyoscyamus, belladonna, and other narcotic sedatives, may be employed in succession. A belladonna plaster worn over the præcordial region appears frequently to exert a happy effect. Opium is admissible, in some cases, bearing in mind the risk of becoming accustomed to its use, and the formation of a habit which is with difficulty broken, and which entails evils of no small magnitude. Palliative means in these, as in other cases, are, of course, to be conjoined with measures which are designed to be curative.

Paroxysms of palpitation characterized by violence and irregularity of the heart's action, may be shortened and mitigated by palliative measures.

Cases of functional disorder often come under the observation of the physician, for the first time, under these circumstances. He is frequently summoned in haste, and finds, perhaps, the patient and friends in a state of great alarm. The first point is to give assurance of absence of danger, so soon as it is ascertained that the disturbance is merely functional. A full opiate affords often the quickest and most reliable method of procuring relief. Revulsive applications are serviceable, such as sinapisms to the chest, or,

compresses moistened with strong aqua ammoniæ. These may be applied over the spine if there be tenderness on pressure. Warm, stimulating foot-baths are highly useful. These measures relieve by obviating the tendency to the accumulation of blood within the heart. This tendency is shown by coldness of the surface and extremities during paroxysms of palpitation. Painful stimulation of the surface is also useful by diverting the attention of the patient from the heart. If an opiate be not employed, the remedies called antispasmodic are indicated, such as the ethereal preparations, the compound spirits of lavender, the aromatic spirit of ammonia, valerian, assafœtida, etc. Some of these may be given in conjunction with, or in addition to opium or the salts of morphia.

In paroxysms characterized by feebleness of the heart's action, with intermittency and a tendency to syncope, prompt relief is often afforded by alcoholic stimulants. Brandy or some form of spirit should be given pretty freely, and not much diluted. Anodynes, antispasmodics, and revulsive applications may be added.

The symptoms associated with palpitation, exclusive of those referable to the heart, must influence, to some extent, palliative measures. Thus, if plethora be manifestly present, a small venesection or local bloodletting by cupping or leeching, may be indicated with a view to immediate relief. Marked coldness of the surface, on the other hand, and prostration, point to the free use of stimulants. Remedies addressed to the stomach, in certain cases, are effectual. If the stomach be distended with gas, carminatives sometimes act indeed like a charm. If acidity or cardialgia are present, an alkaline or antacid remedy, and especially ammonia, may cut short the paroxysm. If the bowels be constipated and flatulent, an active cathartic, or a large, stimulating enema may prove equally efficient.

In paroxysms occurring in persons of a gouty habit, it has been advised to make irritating applications to the joints usually affected, in order to solicit the local manifestations of the disease in these situations. Preparations of colchicum and guaiacum are considered as indicated by palpitation occurring under these circumstances. The palliative measures, however, suited to other cases are applicable, and especially remedies addressed to the stomach.

CHAPTER X.

DISEASES OF THE AORTA.—THORACIC ANEURISMS.

Acute aortitis—Subacute or chronic aortitis—Morbid deposit on the surface of the lining membrane of the aorta—Atheroma—Calcareous deposit—Dilatation of the ascending aorta—Thoracic aneurisms—Definition—Varieties—Anatomical relations, etc.—Causes—Terminations—Symptoms—Physical signs—Diagnosis—Treatment.

Affections of the aorta do not, strictly, fall within the scope of a treatise on the diseases of the heart; but they are with propriety included, not merely on account of the close anatomical relations of the parts affected, but because diseases in these two situations are often associated, and, without proper knowledge and care, certain symptoms and signs due to aortic affections, are liable to be attributed to the heart. The aorta may be the seat of inflammation, acute and chronic, and of structural changes, either resulting from inflammation, or non-inflammatory; it is subject to alterations in calibre, viz., contraction or dilatation, and aneurisms occur oftener in this than in any other portion of the arterial system. This chapter will be devoted to a brief consideration of these several forms of disease, treating of them only so far as they are of interest to the practitioner of medicine.

Acute inflammation of the aorta is one of the most infrequent of diseases. Only a small number of well-marked cases are on record. It might naturally be presumed that the inflammation would be likely to extend into this artery, from the left ventricle, in endocarditis, but it is quite otherwise; the latter disease is sufficiently common, and in the few cases of aortitis which have been observed, it does not appear to have been always either preceded or accompanied by endocardial inflammation. According to Rokitansky, and other late pathologists, the primary seat of inflammation affecting the arteries is in the outer, or, as commonly called, the cellular coat. The middle and lining coats, not containing bloodvessels, are supposed never to be the point of departure of inflammation, but to become involved secondarily in the inflammatory processes.

Vascular engorgement is presented only in the cellular coat. Inflammatory exudation, and pus, either infiltrated or collected in small abscesses, may be here found. The middle and lining coats are thickened softened, friable, easily detached from each other, and presenting sometimes appearances resembling ulcerations. The redness which these coats may present is, probably, always due to the imbibition of the serum of the blood holding in solution hæmatin. Solid deposits are formed within the vessel, consisting chiefly of coagulated fibrin, or decolorized clots. Whether exuded lymph may permeate the middle and lining tissues and accumulate within the artery, is a point on which pathologists are not agreed. These deposits, in proportion to their quantity, must diminish the calibre of the vessel, and obstruct the free passage of blood. They may be sufficient to cause occlusion of some of the arterial branches. Carried onward in masses, or in disintegrated particles, with the current of blood, they may occasion obstruction of remote vessels, and interrupt the circulation in certain portions of the body, possibly leading, thereby, to gangrene.

Acute aortitis is not attended by symptoms distinctive of the disease; or, at all events, if the clinical history embrace any peculiar symptomatic phenomena, they are yet to be ascertained. Disease of the lungs or heart coexisting in most cases, the phenomena referable to the surrounding organs render obscure symptoms proceeding from the aortic inflammation. Association with various concomitant morbid conditions will serve to explain the discrepancy in the descriptive history of different cases which have been reported. It is doubtful whether acute pain ever pertains intrinsically to the disease. Dr. Corrigan attributes the occurrence, in some instances, of painful paroxysms resembling those of angina pectoris, to inflammation seated at the origin of the artery, but these have been only occasionally observed, and it is not certain that there was any direct pathological connection with the aortitis. Œdema of the extremities and of the body generally, or anasarca, observed by Bizot, and considered by him as characteristic of the disease, was probably due to coexisting Bright's disease, the latter affection being but little understood at the time his observations were made. Febrile movement, more or less intense, doubtless accompanies acute inflammation in this situation, and is proportionate to the extent of the aorta involved. It is intelligible that if the current of blood through the arteries be obstructed by the accumulation of coagula or lymph, the pulse will be proportionately small. Feeble-

ness of the pulse must also be an effect of the impaired elasticity of the coats of the artery. It is probable that the transportation of fibrinous plugs with the current of blood to the smaller arteries, occurs in some cases, giving rise to effects which have been noticed in a previous chapter under the head of embolia.[1]

As regards physical signs, violent pulsation of the artery is said to be incident to inflammation. It is possible that this sign may be sometimes appreciable in the second intercostal space, close to the sternum, on the right side, where the artery approaches nearest the thoracic walls. But, as an isolated sign, this is not distinctive of aortitis. The artery is much more accessible to palpation in the epigastrium; but violent pulsation in this situation is sufficiently common without involving inflammation. In a case observed by Dr. Parkes, and cited by Walshe, a loud, rough systolic murmur was audible from the third dorsal vertebra quite down to the lumbar region. The production of a murmur in consequence of the presence of deposits of fibrin and lymph within the affected vessel is readily understood; but structural changes, occurring as either effects of inflammation or of non-inflammatory processes, equally give rise to a murmur, so that this can only be considered as a sign of aortitis when developed while a case is under observation, and associated with other phenomena rendering the existence of the disease probable. In short, physical exploration fails in furnishing definite signs upon which much reliance is to be placed.

From the preceding remarks relating to symptoms and signs it follows that the diagnosis of acute aortitis, in the present state of knowledge, is impracticable. Hence a fuller consideration of the disease, including the subject of the treatment, would be inconsistent with the practical character of this work.

The frequency of subacute or chronic aortitis is problematical. If the anatomical changes so often found within the aorta are of inflammatory origin, as some pathologists have assumed, inflammation of a low grade of intensity must be quite common in this situation. In many, if not most, instances, it is probable that these changes are non-inflammatory; but the discussion of the question, how often or to what extent they involve inflammation, would be unprofitable in a practical point of view, and therefore here out of place. Symptoms and physical signs do not furnish the means of recognizing subacute or chronic aortitis during life.

[1] Chapter IV. page 151.

The anatomical changes occurring within the aorta, to which reference has just been made, involve the presence of morbid deposit. In examining the bodies of aged persons after death, it is rare to find the internal surface of this artery entirely free from disease. Changes due to morbid deposit often occur in the middle aged, especially in males; and they are sometimes observed in young subjects. They are found when there had been, during life, no symptoms denoting disease in this situation; but they are important as leading to dilatation, aneurism, and occasionally perforation or rupture. Their agency in the production of certain physical signs is, also, to be borne in mind.

The deposit may be situated upon the free surface of the lining membrane of the vessel, forming membraniform patches, variable in number, thickness, and size, composed of a white, dense substance, sometimes of a cartilaginous firmness, closely adherent to the membrane. This substance may, possibly, in some instances, be lymph which has exuded from the *vasa vasorum* of the outer coat, and permeated the middle and inner tunics of the artery. According to Rokitansky, it is always derived from the blood within the artery, and is either condensed fibrin, or "an excessive deposition of the lining membrane of the vessels." This deposit in some cases extends over the whole aorta, and even into the communicating arteries. It may lead to occlusion of the latter at the points of communication with the aorta. Although this false membrane becomes, after a time, so closely adherent to the surface of the inner coat of the artery that it cannot be removed without bringing away the latter, it is not united by means of an organized attachment, and does not itself take on organization.

A more common form of deposit is that known as *atheroma*. This deposit takes place beneath the lining membrane of the artery. It occurs first in small, isolated points which increase and coalesce, forming patches of greater or less size. The substance of the deposit is soft and even semi-liquid, or more or less hard. It is found, on microscopical examination, to contain oil-globules in abundance with crystals of cholesterine, and, hence, it is considered as constituting a variety of fatty degeneration. It is frequently associated with fatty change of the muscular substance of the heart to a greater or less extent. The presence of this deposit involves more or less softening and looseness of the middle and lining coats of the artery. The atheromatous deposit is the seat of the calcareous matter so often observed in the bodies of those who die after the middle

period of life, not infrequently occurring during, and sometimes prior to, this period. In the progress of time, the patches of atheroma are transformed into plates or masses of a bony hardness. The lining membrane of the artery covering these plates or masses disappears, and they project into the vessel, coming in direct contact with the current of blood. The interior surface of the vessel is in this way roughened; the current of blood is broken, and the elasticity of the artery impaired or destroyed. The projections within the vessel also serve as a nucleus for the deposit of fibrin, and the calibre of the vessel may thus be considerably diminished, causing obstruction to the free passage of the blood. The transformation of atheromatous into calcareous patches, must be considered as a conservative provision for strengthening the affected portions of the artery, diminishing the liability to dilatation and rupture. If, however, the vessel be extensively calcified, and rendered thereby unyielding and inelastic, the circulation is deprived of the force derived from the recoil of the affected portion of the aorta; an additional labor falls upon the left ventricle which, in consequence, is apt to become hypertrophied. Calcified arteries were formerly said to be ossified, and the term ossification is still sometimes applied to this morbid change, incorrectly, inasmuch as the calcareous deposit lacks the characters of bony texture, resembling the latter only in density and chemical composition.

Calcareous degeneration may exist to a considerable extent without giving rise to any notable symptoms of disease. It is found after death to pervade the aorta more or less extensively, especially at and anterior to the arch, having occasioned, during life, no apparent inconvenience. But it may give rise to aortic murmurs which it is desirable to discriminate from those involving lesions of the aortic valves and orifice. With these lesions, affections of the aorta are frequently associated, but the artery may be extensively diseased without the valves and orifice being involved. How is this fact to be determined during life? The friction of the current of blood in its onward course against the inner surface of the aorta roughened by calcareous patches, causes a murmur; and the regurgitant current or retrograde movement of the column of blood, due to the recoil of the arterial coats, may also cause a murmur, even assuming that the semilunar valves are sufficient. We may thus have a systolic and a diastolic murmur, one or both, produced within the aorta, independently of either obstruction or insufficiency at the aortic orifice. It is desirable to distinguish these murmurs, for they

represent lesions which are of little consequence compared with those involving aortic contraction and regurgitation, and, consequently, serious enlargement of the heart. A systolic murmur produced within the artery just above the orifice, may have its maximum of intensity in the second intercostal space close to the sternum, where the artery is nearest the ear of the auscultator. In this respect it does not differ from a murmur produced at the aortic orifice. It is less likely than the latter murmur to extend below the level of the third rib. It is perhaps more likely to be propagated with considerable intensity into the carotids. These differential points, it must be confessed, are not sufficient always for a positive discrimination. The aortic second sound of the heart is to be taken into consideration. This sound is not likely to be notably impaired if the aortic orifice be not the seat of lesions. At all events, an aortic murmur, whether produced at the orifice, or within the artery above, does not necessarily denote a serious morbid condition, when the aortic second sound is unimpaired. A diastolic murmur produced within the artery above the orifice, provided the semilunar valves be sufficient, is heard in the second and third intercostal spaces on the right side of the sternum. It is not propagated downward, as when it is produced by regurgitation through the aortic orifice into the cavity of the ventricle, the valves being insufficient. In the latter case, the murmur is loudest over the body of the heart and is heard often at the xiphoid cartilage, or even still lower. The integrity of the aortic second sound of the heart is important in determining that a diastolic murmur is produced within the artery alone, without involving insufficiency and regurgitation at the aortic orifice. If the aortic second sound be not notably impaired, it may be assumed that the murmur is produced within the artery. It is, however, to be considered that in proportion as the elasticity of the aorta is diminished, the intensity of the aortic second sound will be lessened. It is to be added, that the intensity or quality of a murmur produced within the aorta, is not evidence of the extent or amount of disease. Calcareous patches, few and small, may so roughen the membrane as to develop a loud rasping murmur; and, on the other hand, calcareous deposit may pervade the artery when the murmur is feeble and soft, the physical conditions, in the latter case, being less favorable for the production of sonorous vibrations. Much will depend on the power with which the left ventricle contracts, as regards the loudness and roughness of aortic murmurs; hence, other things being

equal, they are loud and rough in proportion to the amount of hypertrophy of this ventricle which may be present, and the muscular vigor of the heart.

Dilatation of the ascending aorta is a not infrequent result of atheromatous and calcareous disease. The middle and lining tunics becoming softened, attenuated, and the elasticity of the vessel impaired or lost, dilatation takes place from the distending force of the blood-currents propelled forward by the systole of the left ventricle, and backward by the recoil of the arterial coats beyond the affected portion of the aorta. This result is more apt to follow if the left ventricle become hypertrophied. The ascending portion of the arch and the sinuses of Valsalva are the points most apt to yield to the distension. In the latter situation, the dilatation is probably produced more by the retrograde than the onward current, provided the semilunar valves remain sufficient. According to Dr. Bellingham, visible pulsation of the large arteries of the neck and upper extremities, and a jerking or receding pulse, are characteristic of dilatation of the arch of the aorta. These signs have been noticed in a previous chapter as distinctive of lesions at the aortic orifice permitting regurgitation from the artery into the left ventricle. Their significance of dilatation of the aorta, disconnected from aortic insufficiency, must depend on the presence of adequate evidence that the semilunar valves remain sufficient. This evidence consists in the absence of a murmur denoting regurgitation into the ventricle, and the intensity of the aortic second sound of the heart being but little, if at all impaired. It is only under these conditions that the signs can be considered as indicating aortic dilatation. Dr. Bellingham also states that an impulse synchronous with the pulse is perceived when the ear is applied to the stethoscope laid upon the first bone of the sternum. This impulse may be perceived thus by the ear when it is not communicated to the hand with sufficient force to be appreciable. In connection with these signs, a double rough murmur is perceived which is referable to the aorta and not to the aortic orifice. The diagnosis in some cases, as claimed by the author just mentioned, may be made out by means of this combination of physical signs; but in most cases, the diagnostic points are invalidated by the coexistence of lesions at the aortic orifice.

The foregoing diseases of the aorta, although intrinsically important, and involving pathological questions of much interest, have been passed over cursorily, because the diagnosis is generally

impracticable, and, were it practicable, they are, for the most part, not amenable to treatment. Acute aortitis, if ascertained, would call for measures to diminish the intensity of inflammation and the tendency to coagulation of the fibrin in the blood. Subacute or chronic aortitis is always latent, and it is doubtful whether, were its existence determined, therapeutical measures would be of much avail. Atheromatous deposit is not revealed either by symptoms or signs, and is dependent on a diathesis often incident to age, and the removal of which could hardly be expected. Calcareous plates or masses, and dilatation of the aorta, give rise to murmurs which, with proper attention and knowledge, may, in some instances, be referred to the aorta. The latter is the most important practical point connected with the aortic diseases which have been noticed. Murmurs, systolic and diastolic, may be generated by the currents of blood exclusively within the aorta, when the lining membrane is roughened by calcareous matter or other structural changes such as puckering of the membrane, and by alterations in the calibre of the vessel. These murmurs, if incorrectly referred to the aortic orifice, would denote serious lesions, whilst, in fact, the anatomical changes are comparatively unimportant, and perhaps innocuous. This is to be borne in mind, and it is to be determined, if practicable, in individual cases, whether aortic murmurs are produced within the artery or at the aortic orifice. Owing to the frequent coexistence of lesions situated at this orifice with diseases of the aorta, murmurs are often developed in both situations. Under these circumstances, the discrimination is difficult, and comparatively of small importance.

The subject of thoracic aneurisms claims a more extended consideration than the aortic diseases which have been noticed. To this subject, the remainder of the chapter will be devoted.

THORACIC ANEURISMS.

The term aneurism, in its broadest sense, and in accordance with its etymology,[1] is applicable to every species of dilatation of an arterial trunk. It is convenient, however, to exclude cases in which an artery is slightly or moderately enlarged in its whole

[1] *ἀνεύρυσμα*, a dilatation.

circumference, confining the application of the term to dilatations of this description which are considerable in degree, and circumscribed, as regards the extent of the vessel affected. But the term is applied more particularly to dilatations of a portion of the arterial wall, forming a sac or pouch of greater or less size. Writers treating at length of this subject have divided aneurisms into various kinds, according to the form of the enlargement, the integrity, or otherwise, of the several coats of the artery, etc. Thus, if the dilatation affect the entire circumference of the vessel, the dilated portion is in some instances cylindrical, and in other instances fusiform or spindle-shaped; hence a division after these two forms. The division into *true* and *false* aneurisms has been long maintained, in the former the artery being simply dilated without solution of continuity in any of the coats, in the latter the inner and middle coats having been ruptured or destroyed over a certain space. True aneurisms, according to this distinction, present the same number of tunics as the artery in its healthy state, whilst in a false aneurism, the sac or pouch-like dilatation consists, either entirely or in part, of only the external coat of the artery. The breach in the middle and lining tunics may be the point of departure for the aneurismal dilatation. It is so considered by most writers in the majority of cases, but this is denied by Rokitansky, who thinks that the destruction of these coats is generally consecutive to the dilatation. In the instances in which the latter is assumed, the aneurism is said to be *mixed*. A mixed aneurism, then, is one which, being primarily *true*, in its progress becomes *false*. The tumor formed by an aneurismal sac is generally smooth and globular, but it is sometimes oval or conical in shape, and may be rendered irregular by secondary and even tertiary dilatations giving it a mulberry appearance externally, and causing it to present internally a multilocular arrangement. These variations have given rise to other divisions. A curious variety is called the *dissecting* aneurism. In this kind, rupture of the inner and middle coats of the artery first occurs, and the blood, instead of dilating the outer coat so as to form a sac, detaches this coat over a greater or less distance. In an instance reported by Dr. Pennock,[1] the dissection of the coats extended as far as the primitive iliacs. The aorta in this case presented the appearance

[1] Note in Hope on Diseases of the Heart, 1842, p. 402. Several cases are here reported by the editor, Dr. Pennock. Similar cases have been described by Laennec, Guthrie, and others.

of a double cylinder, that situated internally being the aorta proper, communicating directly with the heart, and surrounding this a much larger cylinder communicating with the inner one by a valvular fissure half an inch in length. In some instances the blood which separates the coats, after passing a certain distance, finds its way again into the proper arterial channel through a second opening.

An account of each of the various kinds of aneurism should, of course, enter into a full consideration of the subject; but, directing attention exclusively to aneurisms of the thoracic aorta, and considering these only so far as they are of interest to the diagnostician, the kinds which are distinguished as sacculated are chiefly important in the present connection. Sacculated aneurisms giving rise to symptoms and signs upon which a diagnosis may be based, in the great majority of cases, are either false or mixed aneurisms. With reference to diagnostic phenomena, certain points relating to sacculated aneurisms are to be kept in view. These points are to be noticed prior to considering the symptoms and physical signs.

Sacculated thoracic aneurisms form tumors varying in size from a pea to the fœtal head, but to give rise to phenomena available for diagnosis, the size must be considerable. Their situation determines, to a considerable extent, their symptoms and signs. They are seated oftenest in the ascending aorta, next in frequency at the arch, and less frequently in the descending aorta. Of eighty-seven cases, in forty they were connected with the ascending aorta, in thirty with the arch, and in sixteen with the descending portion.[1] They occur not infrequently at the sinuses behind the semilunar valves, and in this situation rupture takes place before the tumor attains to a large size, in consequence of the cellular coat which exists in other situations being here wanting and its place supplied by the more delicate pericardium. The effusion of blood when rupture takes place is into the pericardial sac, and death is usually produced almost immediately by mechanical compression of the heart. The diagnosis of aneurism in this situation is impossible. Of two cases that have fallen under my observation, in one the patient fell and expired almost instantly while in the act of leaving the hospital after recovering from an attack of pleurisy. In the other instance the person was found dead, having been, up to the time of death, apparently in perfect health. In neither case were

[1] Swett, op. cit., page 544.

there any symptoms pointing to the heart or large vessels as the seat of disease. Situated above the valves, the aneurism may or may not be accompanied by dilatation of the artery at its orifice, so as to render the valves insufficient; or the aneurism may or may not be complicated with aortic valvular lesions. As regards the effects upon the heart, much depends on the existence or non-existence of aortic regurgitation. If regurgitation take place, enlargement of the heart, commencing and predominating in the left ventricle, is sure to follow; but if the aortic valves remain sufficient, the heart by no means invariably becomes enlarged. Aneurisms of the ascending aorta and arch are generally seated on the convex side of the vessel, and the tumor extends from the vessel in a direction to the right, the distance, of course, being proportionate to the size of the tumor. But it is to be borne in mind that the tumor may spring from the concave or the posterior surface of the vessel and extend in different directions, having, consequently, different anatomical relations to the thoracic walls and the organs within the chest. Whatever may be the direction in which the tumor extends, in proportion to its size, it presses upon surrounding parts, displaces them, interfering with the performance of their functions; it gives rise to local inflammation and abnormal adhesions, causes erosion and atrophy of organized structures, and, finally, ends frequently in rupture or an opening produced by sloughing or ulceration, through which the arterial blood escapes either externally or into some internal part.

The anatomical relations of aneurismal tumors must be considered in order to understand the rationale of certain symptomatic phenomena. Arising, in the majority of cases, from the convex margin of the ascending aorta or the arch, contracting adhesions with the thoracic walls, and erosion of the latter taking place, the integuments are at length raised, forming a visible swelling. This swelling or external tumor is generally situated at a point on the right side of the sternum between the clavicle and the fifth or sixth ribs; but it may make its appearance over the first or second bones of the sternum, or in the neck, or below the left clavicle, and in rare instances on the posterior surface of the chest. But before it becomes apparent to the eye, certain effects are apt to be produced by pressure on internal parts. These effects are still more marked when the aneurismal tumor springs from the concave margin of the vessel. The parts exposed to pressure, and giving rise to symptomatic phenomena, are the trachea, the bronchi, the lungs,

the œsophagus, the par vagum and recurrent nerves, the superior vena cava, the thoracic duct, and the pulmonary artery. The effects of pressure on these parts belong among the symptoms of thoracic aneurism. The anatomical relations of aneurismal tumors arising from the descending aorta are different. Pressure on the parts just mentioned does not occur. An external tumor is not developed unless the aneurism become extremely large. They are apt to contract adhesions to the dorsal vertebræ, leading to erosion of the bony structure, and sometimes an opening takes place into the spinal canal. Situated behind the heart, an aneurismal tumor may displace this organ, and, by pressing it forward, render its action on the thoracic walls so strong as to simulate hypertrophy.

In addition to the size of aneurismal tumors, the portion of the aorta from which they spring, and the direction in which they extend, other points are concerned in the production of symptoms and signs. The mouth of the sac varies in size and form in different cases, allowing more or less freedom of the ingress and egress of blood, and either favoring, or otherwise, the force of the current into the cavity. The opening may be smooth, or roughened by calcareous deposit. The artery in the vicinity of the aneurism may be healthy, but oftener it is more or less diseased. The interior of the sac is frequently studded with calcareous plates. The cavity of the sac may be filled with liquid blood; but it often contains solidified fibrin in more or less abundance. This fibrin, deposited in a series of concentric layers, presents a stratified arrangement, the layers nearest the parietes of the sac being the most condensed, decolorized, and dry, and those in proximity to the blood softer, more moist, and reddened with hæmatin. The size of the cavity receiving blood is, of course, diminished in proportion to the accumulation of solidified fibrin. This deposit is favored by the large size of the sac, the smallness of its mouth, roughness of the interior surface, and feebleness of the heart's action. It must be considered as a conservative provision for strengthening the sac and retarding the progressive increase of the tumor. A spontaneous cure is sometimes effected by an accumulation of fibrin sufficient to obliterate the cavity. Masses of fibrin are liable to become detached from within the sac, forming emboli, or plugs, which are carried onward with the current of blood, and, becoming impacted in arteries more or less remote, occasion obstruction of the circulation, and the consequences of a diminished supply of blood to important parts. Obstruction of arterial branches communicating with the

portion of the aorta where the aneurism is seated, is an important effect. This arises from the deposit of fibrin, and from the small size of the mouth of the sac, or the form of the aperture being such as not to allow free passage of blood into the cavity. It may also proceed from the outward pressure of the aneurismal tumor. The carotid and subclavian arteries may in this way be more or less obstructed, and even obliterated, when aneurisms involve the arch of the aorta.

The formation of a thoracic aneurism always involves a pre-existing morbid condition of the arterial coats, and in most instances this morbid condition is connected with the atheromatous deposit. The middle and lining coats becoming softened, distensible, and sometimes destroyed over a certain space, dilatation is produced by the force of the blood-current, and, as a rule, the yielding parts are more and more dilated by the same force. Various circumstances, which are sufficiently obvious, on the one hand favor, and on the other hand retard the progressive increase of the aneurismal tumor. Other things being equal, the increase in size goes on with a rapidity proportionate to the softened, relaxed state of the sac, the freedom of communication with the artery, the power of the heart's action, and the deficiency of layers of fibrinous deposit. These circumstances varying in different cases, the progress of aneurisms is sometimes extremely slow, and in other cases comparatively rapid. The connection of atheromatous disease is to be borne in mind, for its existence in portions of the artery not involved in the aneurism may give rise to arterial murmurs, which have been already considered; and this disease is apt to be associated with valvular lesions and cardiac enlargement, irrespective of the effects of the aneurism on the heart.

The primary causes of aneurism affecting the aorta are those involved in the production of disease of the coats of this vessel. The supposed agency of muscular exertions or strains in certain cases, irrespective of disease of the artery, may fairly be doubted. An influence apparently belongs to age and sex. Males are far more subject to the affection than females, and it is rare that it occurs prior to the age of thirty or after the age of sixty. These facts are explained by the more frequent occurrence of disease of the coats of the artery in males than in females, by the infrequency of its occurrence prior to the age of thirty and by the rigidity of the arterial walls and lessened power of the heart after sixty. The fact that the ascending aorta and the arch are especially apt to be the

seat of atheromatous disease, explains, in a great measure, the liability of these portions of the aorta to become affected with aneurism. Aneurisms seated in the smaller arterial trunks are frequently of traumatic origin, but it is obvious that aneurisms of the aorta are never attributable to wounds of this vessel. It is possible that hypertrophy of the left ventricle may contribute to the formation of aneurism, especially when seated in the ascending aorta and arch, in consequence of the abnormal force with which the blood is driven into the artery.

The terminations of thoracic aneurisms may be briefly noticed, before entering on the consideration of the symptoms, signs, and diagnosis. A fatal result occurs sooner or later in the vast majority of cases. The different modes in which this result is brought about are of historical rather than practical interest, since they are influenced but little, if at all, by remedial interference.

Eventually, in a large proportion of cases, the aneurismal sac opens, and the patient dies from hemorrhage. But in some instances, pressure on important parts, viz., the trachea, bronchi, lungs, œsophagus, spinal cord, vena cava and pulmonary artery, occasions death before rupture takes place. Death may sometimes be attributable to emboli detached from within the sac. It is needless to say that the existence of aneurism does not preclude the development of various intercurrent affections which may destroy life. The rupture of the aneurismal sac takes place in different situations. It occurs within the pericardium, as already mentioned, when the aneurism is seated below the attachment of this membrane. It also occurs in this situation, occasionally, when the site of the aneurism is above the attachment of the membrane. The latter occurred in five of seventy-nine cases analyzed by Mr. Crisp and Dr. Swett. The relative frequency of rupture in other situations will be most readily represented by giving the combined results of the statistics furnished by the authors just named. Rupture into the *cavities of the heart* took place in nine of one hundred and thirty-eight cases. Of these nine cases, the rupture was into the right auricle in four, the right ventricle in four, and the left ventricle in one. Rupture into the *pulmonary artery* took place in six of two hundred and seventeen cases. In all of these six cases, the aneurism was seated in the ascending aorta or arch. Rupture into the *vena cava* occurred also in six of two hundred and seventeen cases. Rupture into the *pleural sac* took place in fourteen of two hundred and seventeen cases. Aneurisms of the descending aorta are more likely to open in this situation than

those seated in the ascending portion or the arch. The rupture is oftener into the left than into the right pleural cavity. Rupture into the *lung* occurred in eleven of two hundred and fifty-four cases. Rupture into the *œsophagus* took place in sixteen of two hundred and sixty-two cases. When it occurs in this situation the aneurism is generally seated in the transverse or descending portions of the aorta. Rupture into the *trachea* took place in thirteen of two hundred and fifty-four cases. The aneurisms in these cases were generally seated at the arch. Rupture into a *bronchus* took place in eight of two hundred and fifty-four cases. It occurred oftener in the left than in the right bronchus. Of rupture into the *vertebral canal*, only a single instance is contained among the cases analyzed. Rupture *externally* took place in only eight of two hundred and sixty-two cases. It is thus seen that the instances in which the opening takes place into some internal part, greatly preponderate over those in which the rupture is external; of two hundred and sixty-two cases analyzed, internal rupture took place in one hundred and forty-five. As already stated, rupture is a termination in a large proportion of cases; but the number of cases in which death occurs either from results of the aneurism irrespective of rupture, or from intercurrent affections, is nevertheless considerable. Of two hundred and fifty fatal cases, in ninety-two rupture did not take place. Finally, a spontaneous cure of thoracic aneurism is possible. It can take place in but one way, which has been already mentioned, viz., the obliteration of the aneurismal cavity by means of the deposit of fibrin. Recovery, however, occurs in so small a number of instances, that the possibility of its occurrence is hardly to be taken into account in the prognosis. It is hardly possible after the aneurismal tumor has attained to a large size. After a spontaneous cure has taken place, the sac, filled with fibrin, and remaining attached to the artery, presents the appearance of an extraneous tumor. Obsolete aneurisms were regarded as tumors formed without the artery, by Corvisart and others, prior to the researches of Hodgson. They doubtless undergo considerable reduction in size in the progress of time, from contraction of the contained fibrin.

Symptoms of Thoracic Aneurism.

The symptoms of thoracic aneurism are mainly due to pressure of the aneurismal tumor on the surrounding parts. If the tumor

be small, and so situated as not to contract adhesions with, and press upon, certain portions of the intra-thoracic organs, it may remain latent for an indefinite period. Rupture and sudden death occur not very infrequently, when there had been no symptoms to excite suspicion of the existence of aneurism. Aneurismal tumors, however, frequently give rise to symptoms more or less marked and characteristic, which are referable to the respiratory system and voice, the function of deglutition, the venous circulation, and the arterial pulse.

If the tumor press on the trachea, or a bronchus, so as to diminish considerably the calibre of one or both of these tubes, or encroach largely on the space which the lungs should occupy, embarrassment of respiration may occur, manifested especially when an unusual demand is made on the respiratory function, as in active muscular exercise. The enlargement of the tumor, however, being gradual, the diminished calibre of the tubes, or the displacement of the lung-substance, although considerable, does not uniformly occasion notable want of breath even on exercise. But in some cases dyspnœa is a very prominent symptom. In an instance which came under my observation, in which the aneurism was of large size, and seated at the arch, the patient suffered extremely and constantly from labored breathing, being unable to lie down for weeks before death. The most comfortable position was leaning far forward, with the arms resting on the knees; raising the body to the erect posture increased, in a marked degree, the dyspnœa. In this case, no difficulty of breathing was experienced, and the patient was able to perform hard manual labor up to the time when an external tumor became visible. Dyspnœa, however, proceeds from so many and various morbid conditions, that, alone, it is in nowise distinctive of aneurism. Its significance is derived from concomitant signs showing the existence of an intra-thoracic tumor which is probably aneurismal. This remark is also applicable to cough, which in some cases of aneurism is a prominent symptom, being either dry and spasmodic, or accompanied by more or less mucous, and occasionally bloody, expectoration; while in other cases it is slight, and may be wanting. Aneurisms seated at the arch are most apt to be attended by symptoms referable to the respiratory system. The dyspnœa and cough are, in general, effects of mechanical compression of the air-tubes and pulmonary organs; but irritation or stretching of the par vagum and phrenic nerve on the left side may contribute to the development or prominence of these symptoms,

and sometimes give rise to them independently of pressure of the tumor on the trachea, bronchi, or lungs. Dyspnœa and cough, when produced mechanically, are generally attended by wheezing or stridulous breathing, which may be audible at a distance from the patient. This will be noticed presently, in connection with the physical signs of aneurism.

Impairment of the voice, and aphonia, are symptoms which in some cases are highly significant. These symptoms are developed when the tumor involves the recurrent nerve so as to interrupt its functions. This is apt to occur if the aneurism spring from the left side of the transverse portion of the arch. Dependent on the situation of the tumor being such as to occasion pressure, with irritation, of the recurrent nerve, these symptoms characterize in a striking manner certain cases, while in other cases the voice remains unaltered, notwithstanding dyspnœa, cough, and stridulous breathing may be present. Hoarseness, feebleness, or extinction of the voice, if aneurism be not suspected, may lead the practitioner to infer the existence of laryngitis. The coexistence of the pulmonary symptoms just mentioned may appear to sustain this inference. Tracheotomy has been repeatedly performed under these circumstances. In fact, inflammation of the laryngeal mucous membrane and œdema of the glottis are sometimes produced by pressure of the aneurismal tumor; but the alteration and loss of voice may be entirely functional. Dr. Stokes has indicated a point of distinction between functional affection of the voice arising from pressure on the recurent nerve, and the alteration dependent on laryngeal disease, viz., in the latter the hoarseness or aphonia is constant, and in many cases the voice is never restored, while in the former remarkable variations in the tone and power of the voice frequently occur within short spaces of time. In a case of aneurism of the innominata, in which the recurrent nerve was found stretched over the tumor like a broad ribbon, the variations of voice were truly remarkable. "Within twenty-four hours it would change from the highest treble to a deep bass; at one time it was an inaudible whisper, at another hoarse and croaking; and this variability continued up to the period of death."[1] The hoarseness or aphonia due to aneurism may disappear for a time, and again return. This occurred in a case which came under my observation. In the case now referred to, the alteration of the voice was the first symptom

[1] Op. cit., p. 585.

which indicated the existence of any disease. The patient supposed that he had taken cold, and came to the hospital to be treated for an affection of the air-passages. Up to that time he felt no inconvenience in performing active manual labor. Bulging at the top of the sternum, and abundant evidence of an aneurismal tumor pressing on the left bronchus and suppressing respiration in the right lung, were apparent on an examination made some weeks afterwards.

Pressure of the tumor on the œsophagus interferes with the function of deglutition, giving rise to dysphagia from obstruction. This is liable to occur when the site of the aneurism is at the transverse or descending aorta. It is, however, less frequent than the symptoms referable to the respiratory system. It may coexist with the latter, but is sometimes present without them. It may be the only prominent symptom, and, if aneurism be not suspected, stricture of the œsophagus will then be likely to be inferred.

As a judicious precaution, an examination should be made for the signs of aneurism in cases of dysphagia dependent on obstruction seated below the pharynx, before resorting to the use of the probang, since rupture of the aneurismal sac has been produced by the passage of this instrument. The difficulty of deglutition varies, of course, according to the amount of obstruction; it may be slight, or the ingestion of solid food may be impossible, so that the body suffers from inanition. When the obstruction is extreme, the attempt to swallow, especially solids, frequently provokes paroxysms of pain and spasm, together with cough and dyspnœa, followed by regurgitation of the food arrested in its progress down the œsophagus. The patient refers the seat of the difficulty to the top or middle of the sternum, and sometimes to the epigastrium. Dr. Stokes cites a case reported by Dr. Law, in which the patient could not swallow in the recumbent position, but always took his food while sitting up, with the body bent forward and to one side. The explanation of this is sufficiently obvious. The dysphagia has been observed to diminish and even disappear as the aneurism increased in size, a fact to be accounted for by supposing that with the lateral extension of the tumor, the direct pressure on the œsophagus was lessened.[1] The degree of difficulty may be pretty uniform, or it may vary much at different times, owing to variations in the amount of distension of the aneurismal sac, or to the development of spasmodic action in addition to the pressure.

[1] Bellingham, op. cit., p. 594.

Pressure on the superior vena cava, or the venæ innominatæ, gives rise to venous congestion of the face, neck, and upper extremities. The veins of the neck on one or both sides are frequently distended and tortuous, giving rise, in some instances, to a varicose appearance. The face may be congested to such an extent that it presents a deeply livid and swollen aspect. The neck is sometimes puffed out by vascular turgescence and œdema, forming what Dr. Stokes calls a "tippet-like swelling." The distension of the veins, the lividity and œdema, may extend to one or both of the upper extremities. Venous congestion and œdema thus limited, point to obstruction seated, not at the centre of the circulation, but in the venous trunks which have been mentioned. These veins are likely to become involved in aneurisms springing from the ascending and transverse aorta. Absence of jugular pulsation is a point distinguishing the congestion due to obstruction seated above the heart, from that arising from cardiac lesions which involve tricuspid regurgitation. Physical exploration, perhaps, shows that, in connection with notable congestion apparent only above the heart, cardiac disease is either slight or wanting, and thus affords additional evidence of the seat of the obstruction. Under these circumstances, the existence of an aneurismal or other intra-thoracic tumor pressing on the veins which return the blood from the head and upper extremities, is almost certain. Venous congestion, as just described, is by no means present in all cases of aneurism. Like the other symptoms, its absence is not proof that aneurism does not exist; but when marked in the upper portion of the body and wanting below, in conjunction with other symptoms, and with signs pointing to aneurism, it is highly significant.

Inequality in the pulse at the wrists, and the loss of the pulse on one side, are effects of the obstruction of the arteria innominata or the left subclavian incident to certain cases of aneurism. These effects become important symptoms taken in connection with other symptomatic phenomena. The pulse on the left oftener than on the right side is weakened or suppressed, the left subclavian from its situation being the most exposed to pressure. In comparing the pulse on the two sides, it is to be borne in mind that it is normally somewhat more developed in the right than in the left arm; relative weakness on the right side is therefore more significant of disease. The carotid as well as the subclavian artery on one side may be obstructed, so that pulsation in this artery and its branches is relatively feeble or extinct. These effects on the arte-

rial pulse, in general, denote that the aneurismal tumor springs from the arch of the aorta. Owing to a change in the direction of the tumor, arterial pulsation, which had been at one time suppressed in the neck or wrist, may be subsequently restored; and, for the same reason, having been weakened, it may become stronger. In these instances, the weakness and suppression were due to the outward pressure of the aneurismal tumor on the subclavian, carotid, or innominata; but in the instances in which these arterial trunks are obstructed from within by fibrinous deposit, the deficiency or absence of pulsation is likely to remain unaltered.

The symptoms which have been noticed are important in aiding to determine the existence of an aneurism and its probable seat. These symptoms may be present, individually or collectively, in different cases; each may exist without the others, and all may be wanting. Singly or combined, they are not pathognomonic of aneurism. Being mostly the immediate effects of eccentric pressure, they may be alike produced by any intra-thoracic tumor. Hence, their diagnostic value depends on other evidence of the existence of aneurism being conjoined, especially that furnished by physical signs, to be presently considered. Other symptoms less characteristic and consisting of secondary or remote effects remain to be briefly noticed.

More or less pain usually attends the progress of thoracic aneurisms. Pain, however, is less constant and less marked as a symptom in aneurisms seated within the chest than within the abdomen. Aneurism of the abdominal aorta frequently gives rise to intense, persisting pain, while this is true of only a small proportion of cases of aneurism affecting the thoracic aorta. Of thoracic aneurisms, those springing from the descending aorta are far more apt to give rise to pain than those seated in the ascending aorta or at the arch. The pain is especially marked if the aneurismal sac cause erosion of the bodies of the vertebræ. In these cases, patients describe the pain as boring or gnawing in character, and it is sometimes referred to a small circumscribed portion of the vertebral column. Aneurisms seated in the ascending or transverse aorta are often unattended by severe pain, but in some cases it is a prominent symptom. In these cases, it is generally intermittent, and resembles that of a neuralgic affection, being lancinating in character, shifting its situation, and shooting in various directions. In other cases, an obtuse, persisting pain is complained of. The pain may be referred to different portions of the chest,

extending not infrequently to the shoulders, neck, and arms. Occasionally, it bears some resemblance to the pain of angina pectoris. When an external tumor makes its appearance, the pain may be referred to the part where it appears; and prior to this event, pain in some cases is not present. The parts over an aneurismal tumor are often tender, rendering pressure and percussion painful.

Paraplegia becomes a symptom of aneurism, if, when seated in the descending aorta, it leads to erosion of the vertebræ, and the pressure of the tumor falls upon the spinal cord.

Hemiplegia may occur as a remote effect. Clinical observation shows its occurrence in a certain proportion of cases; but it may be dependent on disease of the cerebral arteries, analogous to that which, in the aorta, preceded the formation of aneurism. Under these circumstances, its occurrence may be merely due to coincidence. Exclusive of these instances, it is probably sometimes due to fibrinous coagula detached from within the aneurismal sac and arrested in the arteries of the brain. Dr. Stokes attributes this and other symptoms referable to the brain, to diminished supply of blood, in the cases in which the carotid artery on one side is obstructed. More importance probably belongs to the cerebral congestion incident, in certain cases, to pressure on the vena cava. In connection with the appearances denoting interruption of the return of blood to the heart, drowsiness, dulness of the intellect, obtuse pain in the head, and other signs denoting passive congestion of the brain, are usually present; and in one case under my observation, paralysis of the muscles of the face on one side existed. It is intelligible that the vascular fulness in these cases should favor the occurrence of extravasation giving rise to apoplexy and hemiplegia.

The existence of aneurism does not involve, directly and speedily, any notable change in the general aspect of the patient. Rokitansky states that if the aneurismal sac attain to a large size, sufficient blood may be withdrawn from the circulation to induce anæmia. Patients sometimes preserve their weight and strength to the last, but in other cases both undergo more or less diminution. From an analysis of seventeen cases of aneurism seated at the arch, Dr. Walshe is led to the conclusion that the difference in different cases as regards loss of weight and strength, is mainly owing to the presence or absence of severe pain. In proportion as this element is prominent, patients emaciate and become enfeebled. Extreme

emaciation is sometimes produced by pressure of the aneurismal tumor on the thoracic duct.

Dr. Gairdner, of Edinburgh, and Dr. Banks, of Dublin, have recently called attention to the occurrence of contraction of the pupil of the eye, on the side of the aneurism, when the tumor extends high up in the neck. This effect is attributed to pressure upon the sympathetic nerve in the neck, division of this nerve on one side having been found in experiments on living animals to be followed by contraction of the pupil of that side.[1]

Gangrene of the lung is a rare symptomatic event, which, according to Dr. Carswell, is induced by compression of the nutrient arteries of the lung by the aneurismal tumor.[2]

Physical Signs of Thoracic Aneurism.

The physical signs of thoracic aneurism are furnished by Inspection, Palpation, Percussion, and Auscultation. I shall consider the phenomena obtained by these methods, severally, following the order in which they are enumerated.

Inspection is frequently not available until the aneurismal sac presses upon the parietes of the chest at some point and gives rise to visible bulging of the surface. The presence of an external swelling or tumor is determined by the eye. At first slight, and limited to a circumscribed area, the swelling may increase so as to form a tumor as large as the fœtal head. The form is usually conical, and the surface, if the tumor be of considerable size, is smooth, and frequently presents a glazed appearance. Pulsatory movements at the site of the bulging, or tumor, may or may not be apparent on inspection. A pulsation may be seen when bulging has not occurred. The appearance is then, as remarked by Dr. Stokes, as if two hearts were beating in the chest in different situations. This pulsation without swelling may sometimes be discovered by looking across the surface with the eye brought down to a level of the chest, when it is not apparent, if the ordinary mode of inspection be alone employed. This is a practical point to be borne in mind.

Palpation enables the observer to ascertain the form of the swell-

[1] Bellingham, op. cit., page 609.
[2] Stokes, op. cit., page 587.

ing, the condition of the surface as regards smoothness, and the amount of resistance to pressure. By manipulations with the hand, also, perforation of the thoracic walls may be ascertained; the edges of the eroded ribs or sternum at the aperture through which the aneurismal sac protrudes, may be felt. Fluidity of the contents of the tumor is sometimes apparent to the touch. The liquid contents of the sac may be diminished by manual pressure, and reduction of the hernia-like protrusion perhaps effected. Much compression of the tumor, however, is not to be advised, since there may be risk of producing rupture, or of detaching coagulated fibrin from within the sac, and thus giving rise to emboli, as well as weakening the aneurismal walls.

Palpation is especially useful in determining the presence and character of pulsatory movements. Assuming that bulging exists, pulsation constitutes important evidence of its being aneurismal. As a rule, an external aneurismal tumor is pulsatile; but to this rule there are exceptions. If the sac be nearly or quite filled with solid fibrin, the stream of blood through the artery small, and the heart's action weak, an impulse, visible or tactile, may be wanting. On the other hand, intra-thoracic tumors not aneurismal, often present distinct and strong pulsation. The impulse varies greatly in strength in different cases, being sometimes extremely powerful, raising with force the head applied for auscultation, and accompanied by a shock which agitates the whole body, and of which the patient is painfully conscious; in other cases it is scarcely perceptible, and between these extremes every degree of gradation may be observed. The character of the aneurismal impulse, when strong, as Dr. Stokes justly remarks, differs from that of the heart's beat in the state of health or of active hypertrophy. The difference arises from the fact that in aneurism the impulse is due to the momentum communicated to a column of liquid, while the beat of the heart is owing to the pressure of the apex of the organ against the chest in its elongating and rotating movements. Quoting the language of the author just named: "The aneurismal beat generally gives the idea of a forcible blow, having a force equal in all directions, while that of the heart conveys the sensation of a mobile but solid body, which, in many instances at least, presents its greatest force at a particular point."[1] This character of impulse, and its strength, are proportionate to the proximity of the sac to the in-

[1] Op. cit., p. 554.

tegument; the deficiency of solid fibrin, or, in other words, the relative amount of liquid blood within the aneurismal cavity; the freedom of communication between the aneurismal cavity and the artery, and the power of the heart's action. The impulse may be single or double. When single, it is synchronous with the ventricular systole, being due directly to the contraction of the left ventricle. If double, the second impulse must be produced by the recoil of the arterial coats following distension of the artery by the onward current of blood; it is thus indirectly due to the contraction of the left ventricle, and coincides in time with the second sound of the heart, or, in other words, it is diastolic. A double pulsation, therefore, consists of a systolic and a diastolic impulse.

In these remarks it is assumed that the aneurismal sac has led to bulging or an external tumor. But, impulse may be perceived by the touch, as well as by the eye, before bulging is apparent. A throbbing, synchronous, or nearly so, with the systole of the heart, may be felt, over a circumscribed space, at a point more or less removed from the seat of the apex-beat. There appear to be two hearts beating within the chest. The presence of the apex-beat at or near its normal situation, is evidence that the abnormal pulsation is not of the heart itself; and that it is not the apex-beat propagated at a distance from the apex, is shown by its being felt within a circumscribed space, and not felt between this space and the point where the apex comes into contact with the parietes of the chest. A source of fallacy connected with the heart may be here mentioned. Free regurgitation through the tricuspid orifice with dilatation of the right auricle and hypertrophy of the right ventricle, may occasion a strong pulsation on the right side of the sternum. An instance related by Dr. Stokes has been referred to in a former part of this work.[1] Aneurismal pulsation without bulging, varies in different cases from an intensity exceeding considerably that of the heart's beat, to a feebleness so great that it is almost imperceptible. The throbbing may sometimes be perceived in some cases by placing one hand on the posterior part of the chest, and making firm pressure with the other hand over the upper part of the sternum, when, with a manual examination by one hand alone, it is not appreciable. The pulsation is most evident or marked at the end of an expiratory act. If the aneurism be seated at the arch and extend upwards, a pulsation may be felt

[1] *Vide* p. 54.

at an early period, before bulging occurs, by passing the finger into the sternal notch, and pressing downward towards the artery. The aneurismal tumor may sometimes be felt in this way, before any visible swelling occurs.

Tactile fremitus, or thrill, with the heart's systole, felt over an aneurismal tumor, is a physical sign which in some cases is strongly marked, but it is by no means uniformly present. It may be present when the impulse is slight or wanting. It is sometimes present in cases of intra-thoracic tumor, not aneurismal. While its absence is not proof that a tumor is not aneurismal, and when present, it does not constitute positive evidence of aneurism, it has, nevertheless, in conjunction with other signs, considerable value, especially if it exist in a notable degree.

Percussion is useful in certain cases by aiding to determine the existence of a tumor, and affords evidence of its size. If an external tumor have formed, its size, as determined by the eye and touch, is no criterion of the size of the aneurismal sac; the latter extends over an area, greater or less, without the limits of the visible tumor. Its extent may sometimes be ascertained with considerable accuracy by means of percussion, provided the pressure on the lung-substance has not induced induration, in which case the percussion-dulness will extend farther than the walls of the sac. It is to be borne in mind that the lung may cover a portion of the aneurismal sac, so that the limits of the latter are not defined by absence of pulmonary resonance. An aneurismal sac, in fact, like the heart, has its area of superficial, and its area of deep dulness on percussion. The former marks the space over which the sac is uncovered of lung; and the latter the distance to which the sac extends beneath the lung. The situation of the dulness goes to show the probable origin of the tumor. Well defined abnormal dulness over the site of the ascending and transverse aorta, in connection with other signs and with symptoms, points to the existence of aneurism. An aneurismal tumor of small size in the situations just named, if not arising from the posterior margin of the vessel, may occasion an abnormal degree and extent of dulness determinable by careful percussion. Dr. Walshe states that "a sac as large as a good-sized walnut may be discovered, if it lie anywhere between the second right interspace and the left border of the sternum, and there be no special and unusual source of difficulty in the way." Percussion is less available if the aneurismal sac spring from the descending aorta. When the sac attains to a certain size, however, it gives

rise to dulness sufficiently defined behind in the interscapular space. It is obvious that percussion, alone, can only furnish evidence that abnormal dulness from some cause exists; that this dulness depends on an aneurism, and not on an intra-thoracic tumor, not aneurismal, or other causes, is to be determined by other signs and by symptoms.

The signs furnished by *auscultation* are referable not alone to the aneurismal sac, or the artery with which it communicates, but to the trachea, bronchi, and pulmonary organs. Directing attention first to the signs proceeding directly from the aneurism, it gives rise, in a certain proportion of cases, to a bellows murmur. This sign is by no means constant; it is frequently wanting. Dr. Stokes, indeed, regards it as an accidental phenomenon so commonly wanting that he considers its occurrence as exceptional. Its infrequency is exaggerated in this statement; but, owing to its being so often absent, it has far less value, as a sign of thoracic aneurism, than has been generally supposed. As an isolated sign, it possesses very small value for other reasons than its want of constancy. Aortic murmurs are sufficiently common, exclusive of aneurism. It has been already seen that they are often generated by the passage of the blood-current over the internal surface of the vessel, when roughened by atheromatous and calcareous disease, without dilatation. Alone, therefore, a bellows murmur referable to the aorta is not proof of the existence of aneurism. Other signs and symptoms must be conjoined to render it significant of this affection. It can hardly be said to contribute evidence of aneurism unless the existence of a tumor be determinable, and even then it is not distinctive, since an intra-thoracic tumor, not aneurismal, by pressure on the aorta, may give rise to murmur. The passage of blood within and without an aneurismal sac does, however, give rise to murmur in a certain proportion of cases. This murmur varies in intensity from the faintest puff to a loudness exceeding that of the most intense cardiac murmurs. It may be soft or rough. It is sometimes most marked over the most prominent portion of the aneurismal tumor, and in other instances it is heard loudest at the base of the tumor. It is said to be, as a rule, less prolonged than valvular murmurs and lower in pitch. Aneurismal murmur may be single or double. If single, it is usually, but not invariably, systolic, *i. e.*, synchronous with the ventricular systole. The second murmur is synchronous with the second sound of the heart, and may, therefore, be distinguished as diastolic. The latter

is rarely, if ever, rough, and is less intense than the systolic murmur. An aneurismal murmur is to be discriminated from cardiac and arterial murmurs. A murmur emanating from the heart may be propagated to the aneurism. A propagated cardiac murmur may perhaps be more intense over the aneurism, if the tumor be near the surface, than at any point between the aneurism and its source; but its maximum of intensity will be at or just above the base of the heart. If more intense over the aneurism than at or near the base of the heart, it may be considered as not cardiac in its origin. But, in most cases, if a cardiac murmur coexist with an aneurismal, comparison of the two murmurs will show differences as regards quality and pitch, sufficient to denote that they are distinct from each other. A murmur propagated from the heart must be produced at the aortic orifice. A mitral murmur is not conducted along the aorta; hence, when a coexisting cardiac murmur is ascertained to be mitral, its identity with a murmur heard over an aneurismal tumor is not a matter of question. Murmur emanating from the artery, elsewhere than at its point of communication with the aneurism, may be due to disease of the arterial coats, or it may be inorganic, *i. e.*, dependent on blood-changes. If due to the former, the maximum of intensity of the murmur will not be likely to be at the aneurismal tumor; if to the latter, the murmur will generally be diffused over the vessels of the neck and attended by venous hum. An anæmic condition doubtless favors the production of an aneurismal murmur and enhances its intensity. Finally, an aneurismal murmur is found to vary at different periods, and even from day to day; and it may exist for a certain period with marked intensity, so as even to be appreciable without auscultation, and subsequently disappear.[1]

Aneurismal sounds, as well as cardiac, are to be distinguished from murmurs. A double sound, corresponding to the systolic and diastolic sound of the heart, is usually heard on auscultation of an aneurismal tumor springing from the aorta. The two sounds resemble those of the heart, not only in rhythm, but in other characters. Dr. Stokes remarks: "They are so similar to those of the heart, that, were a good observer blindfolded, and the stethoscope placed for him over the seat of the disease, he would find it difficult, if not impossible, to distinguish them from the ordinary sounds of an excited heart." I believe them to be neither more

[1] Walshe, op. cit., page 749.

nor less than the heart-sounds transmitted by the walls of the artery and the current of blood, to the aneurismal sac. Holding this belief, I do not deem it necessary to discuss the modes in which the sounds have been supposed to be produced within the sac. That they should be propagated so as to be heard with more intensity over the aneurism than at any point between it and the heart, is readily conceivable in view of the nearness to the ear of the arterial walls and current of blood, when the aneurismal tumor is auscultated. The systolic sound appears to be sometimes reinforced by an element of impulsion derived from the shock communicated to the sac by the onward current of blood; and this element of impulsion is sometimes the only sound appreciable. Either the systolic or diastolic sound may be heard, to the exclusion of the other, and both may be wanting. The latter is likely to occur under the same combination of physical circumstances which renders an impulse extremely feeble or inappreciable.

The auscultatory signs referable to the trachea, bronchi, and pulmonary organs are due to compression of these parts by the aneurismal sac. These signs may be present before, as well as after, the appearance of an external tumor. They are of considerable importance in determining the existence and seat of aneurism.

Pressure on the lower part of the trachea gives rise, as already stated, to wheezing or stridulous breathing, which may sometimes be perceived at a distance from the patient. If the voice be not affected, it is evident that it proceeds from a point below the larynx. But if there be room for doubt, the stethoscope indicates that it is from below. It may be produced in the bronchus on one side before the aneurismal tumor ascends sufficiently to press upon the trachea. It may be perceived, especially if produced in a bronchus, and referred to its seat, by means of auscultation, when it is not loud enough to be apparent without the aid of the stethoscope. Dr. Stokes distinguishes this sign as "*stridor from below.*" It is obvious that it may be produced by a tumor of any kind making pressure on the trachea or bronchi. The practical point is to determine, by auscultation, that a wheezing sound, either audible at a distance, or heard with the stethoscope only, is produced, not at the larynx or the upper part of the trachea, but below, at the bifurcation or in a bronchus. If dyspnœa or labored respiration exist, the seat of the obstruction is thus ascertained. The existence of a tumor pressing upon the air-tubes in this situation is rendered highly probable,

and it remains for the aneurismal character of the tumor to be shown by other signs.

An aneurismal tumor may compress a bronchus so as to diminish, and even suppress, respiration in one lung. Absence of the respiratory murmur on one side, or a marked disparity in the intensity of the murmur between the two sides, may thus become important signs of the existence and degree of bronchial obstruction. The significance of these signs depends, of course, on the absence of causes of diminished or suppressed respiration on one side, other than occlusion or narrowing of a bronchus by the pressure of a tumor. Bronchitis, emphysema, pleurisy with effusion, and the presence of a foreign body, are to be excluded. That the obstruction is not due to enlarged bronchial glands, or intra-thoracic tumor not aneurismal, is to be determined by other signs pointing to the existence of aneurism.

Finally, absence of the respiratory murmur and of vocal resonance over a circumscribed space, or around an external tumor, concurs with the evidence afforded by percussion, either in rendering probable the existence of a tumor within the chest, not apparent to the eye, or, if visible, in determining the space within the chest which the tumor occupies, to the exclusion of the pulmonary organs.

Diagnosis of Thoracic Aneurisms.

A reviewal of the preceding pages will show that the symptomatology of thoracic aneurism furnishes nothing exclusively distinctive of this affection; that is to say, there are no symptoms or signs which are individually pathognomonic. An impulse over a circumscribed space at certain points, distinct from the apex-beat, or a pulsating tumor, renders the presumption strong that aneurism exists; but additional evidence is necessary for a positive diagnosis. Even if bellows murmur and thrill are added, the existence of aneurism is not unquestionable. The diagnostic force of these signs depends considerably on their degree of prominence. If they be combined and strongly marked, the chances against aneurism are small. If, in addition, the point at which an impulse or a pulsating tumor is observed correspond with the situation in which an aneurism springing from the aorta may be expected to be discovered; and if percussion and auscultation show the presence of an intra-thoracic

tumor which can be traced in a direction toward the aorta, there is scarcely room for doubt as to the diagnosis. These diagnostic points are by no means always available. An aneurism may be so situated as regards the thoracic walls, that an impulse is not appreciable, or, if there be a tumor, it may not pulsate, nor present either murmur or thrill. On the other hand, a tumor not aneurismal, may present murmur, pulsation, and thrill. The situation of the tumor is a point of considerable importance. Bearing in mind that, in the great majority of cases, aneurism springs from the ascending or transverse portion of the aorta, and makes its appearance externally either to the right of the sternum, or at the upper or middle portion of the sternum, or to the left of this bone, a tumor thus situated is likely to prove to be aneurismal. If not aneurismal, it is probably a carcinomatous mediastinal tumor. The latter lying over the aorta may pulsate pretty strongly, and by pressing on the artery may develop a bellows murmur. Carcinomatous disease, however, in this situation is a much rarer affection than aneurism, so that the chances of the latter preponderate. Age and sex are to be taken into account. A tumor occupying a site in which an aneurism is apt to make its appearance, is more likely to prove to be aneurismal if the patient be a male and between the ages of thirty and sixty. In general, with proper knowledge and care, taking into view all the circumstances of the case, if an aneurismal tumor be apparent externally, its character may be ascertained without great difficulty, and the diagnosis made with positiveness. It is hardly necessary to notice the differential points which distinguish an aneurismal tumor from pulsating empyema, or pericarditis with effusion, for the existence of these affections is readily determined by their proper diagnostic characters.

An aneurism not in contact with the thoracic walls so as to give rise to an impulse appreciable by the eye or touch, and not forming an external tumor, offers a more difficult problem in diagnosis. Certain symptoms which have been considered, viz., embarrassed or stridulous breathing, dysphagia, venous congestion of the face and upper extremities, and inequality of the pulse on the two sides, should excite suspicion of aneurism, especially if they are found in combination, and, also, individually, provided they are not obviously referable to other morbid conditions. Physical exploration, under these circumstances, is essential, not alone in developing signs which point directly to the existence of aneurism, but by showing that other morbid conditions which would account for the symp-

toms, do not exist, and thus leading to a diagnosis, indirectly, by way of exclusion. The chest is to be carefully examined with reference to cardiac disease, since the symptoms and physical signs of aneurism of the ascending and transverse aorta have many points in common with those referable to the heart. The absence of cardiac murmurs and of enlargement of the heart warrants the conclusion that the symptomatic phenomena are not due to lesions of this organ. But even if the heart be not altogether free from disease, it may be sufficiently clear that certain symptoms and signs are not of cardiac origin. Thus, a mitral regurgitant murmur is easily distinguished from a murmur referable to the aorta; and the amount of enlargement of the heart may be obviously inadequate to account for the disturbance of the respiration and circulation. Auscultation of the lungs and air-passages is to be employed in order to ascertain whether the respiratory murmur be diminished or suppressed on one side from compression of a bronchus, and whether the respiratory sound in the larynx and trachea be pure. If stridulous breathing be heard, with or without the stethoscope, this instrument will show the point at which it is produced, and thus indicate the seat of the obstruction. Percussion and auscultation conjoined are to be resorted to in order to ascertain whether there be not dulness, together with absence of the respiratory murmur and vocal resonance, within a circumscribed space, so situated that the presence of a tumor having relations to the aorta similar to those which aneurisms are known to have, is rendered highly probable or almost certain. A bellows-murmur either localized in this circumscribed space, or heard here with maximum intensity, and the heart-sounds abnormally transmitted to the same space, are signs entitled to a certain amount of weight. Availing himself of the diagnostic points just recapitulated, the diagnostician may be able to decide on the existence of aneurism, not with absolute certainty, but with much positiveness. The liability to error proceeds from the possibility of enlarged bronchial glands, or intrathoracic tumor of some kind, not aneurismal, pressing on the trachea, or a bronchus, the subclavian or innominata, the recurrent nerve, the œsophagus, and the vena cava, so as to give rise to more or less of the symptoms and signs due to the pressure of an aneurismal tumor on these parts. The probability of the tumor being aneurismal is considerably increased if the patient be over thirty years of age and of the male sex.

In connection with the subject of diagnosis, it may not be amiss

to call attention to the importance of examining for a visible impulse with the eye brought to a level with the surface of the chest; and, for a tactile impulse, with one hand applied to the posterior, and the other to the anterior portion of the chest, making firm pressure with the latter hand. On the very day this paragraph is penned, a case has fallen under my observation in which an impulse was rendered apparent to the eye and touch by these methods of examination only. It is true that an impulse is not proof positive of the existence of aneurism, but, even if not strongly marked, its value as a diagnostic sign is considerable. It may be important to caution the young observer against mistaking pulsation of the subclavian artery for an aneurismal impulse. It is to be borne in mind that pulsation of this artery is sometimes visible and felt, and especially in cases of disease of the heart involving aortic regurgitation; but, under the latter circumstances, the carotid and other arteries are observed at the same time to pulsate strongly.

Symptoms and signs pointing to the existence of aneurism, are far less available for diagnosis when the aneurismal tumor springs from the descending aorta, than when it is seated at the ascending portion or arch of the vessel. As regards symptoms, labored and stridulous breathing, aphonia or hoarseness, venous congestion of the upper portion of the body, and inequality of the pulse, are not produced unless the tumor attains to a great size. Dysphagia from obstruction of the œsophagus is liable to occur, and this symptom, when not connected with disease of the pharynx, should always excite suspicion of aneurism. The frequent occurrence of persisting, boring or gnawing pain referred to a particular portion of the spinal column, is to be borne in mind. This should suggest to the mind the possibility of aneurism. The same is to be said of a disposition to keep the body bent forward, in consequence of pain in assuming the erect posture, and of the occurrence of paraplegia, the latter proceeding from erosion of the vertebræ by the pressure of the aneurismal tumor. As regards signs, obstruction of the trachea or a bronchus, and diminished respiratory murmur on one side, are not likely to occur in cases of aneurism seated in the descending aorta. But percussion in the interscapular space may show dulness within a circumscribed area, the limits of which may also be defined by abrupt cessation of respiratory murmur and of vocal resonance. A bellows-murmur may be discovered within this area, not transmitted from the heart, and possibly the heart-sounds may be unduly audible. These signs are not conclusive, but they

point to the existence of aneurism. A positive diagnosis is hardly practicable prior to the development of external bulging or a pulsating tumor. So latent are aneurisms in this situation in some cases that, although symptoms denoting some indefinite ailment have been long experienced, an external tumor may be the first event which excites suspicion of the nature of the disease. Physical exploration is of great value in these cases by enabling the diagnostician to exclude certain affections, for example, chronic pleurisy or empyema, for which, otherwise, the disease might be mistaken.

Treatment of Thoracic Aneurism.

Recovery from thoracic aneurism is possible. The chances against recovery, however, preponderate so vastly that in any given case there is scarcely ground to hope for this result. Still, the possibility of recovery is not to be lost sight of in the management of the cases which it is the misfortune of the physician to meet with in practice. But although recovery is not to be looked for, the progress of the affection may be more or less slow, and the physician may reasonably hope to contribute, by judicious management, to postpone for an indefinite period the fatal result. Moreover, the palliation of distressing symptoms, always an important object of treatment, furnishes scope, in the progress of this affection, for the useful application of remedies.

Recovery from thoracic aneurism can only be effected by the deposit within the sac of successive layers of solid fibrin until the cavity is obliterated, leaving the channel of the artery free. Can this curative process be promoted by medical treatment? There are no medicines which exert a special effect for this end. The general conditions favorable for the process are, an abundance of the fibrinous constituent of the blood, with an equable and not too active state of the circulation. These are conditions which are to some extent controllable. Those which depend on local circumstances, such as the size and shape of the cavity, the form and direction of the aperture, the state of the interior of the sac as regards roughness, etc., are obviously beyond control. The indications for treatment, then, with a view to the possibility of recovery are, to maintain and perhaps increase the relative proportion of the fibrinous constituent of the blood, and to secure regularity

of the action of the heart, obviating, on the one hand, over-excitement, and, on the other hand, undue feebleness. How are these indications to be fulfilled? In brief, by a nutritious, and, in some instances, a generous diet, embracing a good proportion of animal food; by tonic remedies and stimulants when the appetite and digestive powers are enfeebled; and by the restricted use of liquids which, by increasing the quantity of blood, lessen the proportion of fibrin to the mass. These measures relate to the first indication, viz., to maintain or increase the fibrin. The second indication, viz., to secure regularity of the heart's action, is to be fulfilled by avoiding active exercise, mental excitement, and other causes inducing disturbance or undue activity of the circulation; by moderate depletion by means of saline laxatives, or resorting perhaps in some cases to small bleedings if decided plethora exist, but, on the other hand, prescribing ferruginous remedies if anæmia be present; and by the use of sedatives if the circulation be unduly and persistingly over-excited.

These indications, having reference to the possibility of recovery, relate not less to an object of management more likely to be attained, viz., to retard the progress of the affection, and thus postpone the fatal result. This object is alike secured by the deposit of fibrin within the aneurismal cavity, by which the walls of the sac are strengthened, and the tendency to its enlargement thereby lessened, and by tranquillity of the heart's action. The means, then, by which the physician may hope to prolong life are those which, at the same time, afford the best chance, slight as this is, of recovery.

Potent measures of treatment have been heretofore advised and pursued with the expectation of retarding the progress of thoracic aneurisms and with a faint hope of effecting a cure. The plan of treatment introduced about half a century ago by Albertini and Valsalva, adopted by Laennec and Bouillaud, and recommended in a modified form by Hope, obtained more favor and currency than any other. This plan consisted in repeated copious bleedings, as much quietude as possible of body and mind, and reduction of the diet to the lowest point compatible with life. It is unnecessary to show the false basis of this plan, or its pernicious results, since it is now abandoned by all judicious practitioners; nor is it likely that any method involving measures of equal potency, if proposed, would meet with favorable consideration. On this subject, the remarks by Dr. Stokes are so just and forcible that I shall quote

his words: "It is to be doubted whether we are ever justified in adopting any measures which, while they are directed, under theoretical views, to the cure of the disease, materially interfere with the patient's condition. It often happens that a patient who has not been thus interfered with will continue with unimpaired health and strength for a great length of time until he is so unfortunate as to be placed under treatment for the cure of his aneurism. For then all the evils which have been pointed out as occurring in cases of indolent disease of the heart, when injured by ignorant treatment, are induced. The patient's mind becomes excited and apprehensive, his system is weakened by depletion, and his digestive functions ruined by starvation. The forces by which he can resist disease are broken down, his blood becomes uncoagulable, his tissues unresisting. The force of the aneurismal throb is augmented, and a disease which, under other circumstances, might have endured for years with but little interference with the general health, is turned into a rapid and destructive malady."[1]

Pursuing no special plan of treatment, the management must depend very much on the circumstances pertaining to individual cases. Is the patient of a full plethoric habit, the vessels over-repleted, and the heart over-stimulated; depletion may be called for, care being taken not to carry it beyond the point of restoring the blood to its normal condition. Is the patient anæmic, and the heart's action excited by impoverished blood; a nutritious, animal diet, together with preparations of iron and perhaps porter, wine, or spirits, are important, being careful not to push these measures to an extreme, and thereby induce evils equal to those incident to anæmia. Is the appetite good and digestion active; indulgence at the table is to be restrained. On the other hand, is the appetite poor and digestion weak; measures to improve both are indicated. The restricted use of liquids is an important point in the management, conducing, in connection with an appropriate diet, to the formation of blood rich in fibrin, and not excessive in quantity. These ends are promoted by a limited supply of liquid, far better than by the elimination of liquid by means of hydragogue cathartics and diuretics.

A regular and tranquil condition of the heart being highly desirable in all cases, everything which excites unduly this organ is, of course, as far as possible, to be avoided. Active muscular

[1] Op. cit., p. 606.

exercise, mental excitement, the abuse of alcoholic stimulants, etc., are to be interdicted. It is an important question, in this connection, how far exercise can be taken with safety or advantage. Some writers recommend perfect quietude, and enjoining the patient to remain a considerable portion of the time in the recumbent posture.[1] The propriety of this is more than doubtful. The appetite, digestion, and general condition of the body must suffer from such inaction, so that the risk of doing harm by impoverishing the blood and weakening the vital forces, is greater than the liability to injury from moderate exercise. An amount of exercise which can be taken without accelerating the circulation, will be likely to be useful rather than injurious.

An important point in the treatment concerns the mental condition of the patient. A full knowledge of the nature of the affection, and of the accidents to which he is exposed, can hardly fail, in most cases, to induce depression and apprehension, the effect of which must be, to a certain extent, unfavorable. When it can be done without breach of good faith, or a violation of truth, the physician will do wisely in forbearing to enter into an elaborate exposition of the character, tendencies, and results of thoracic aneurisms. While deception here, as in other forms of disease, is unworthy the character of a physician, as well as unjust to the patient, it is fair to present as favorable a view of the case as facts will warrant. The patient may be encouraged with the hope of the affection progressing very slowly, or remaining stationary for an indefinite period, and even with the possibility of recovery. He will thus be spared not only a portion of the unhappiness to which the affection is calculated to give rise, but the unfavorable influence of excessive anxiety and gloom; and he will, moreover, be more disposed to persevere in following faithfully regulations of diet and regimen.

If the action of the heart be habitually excited or irregular, sedative remedies are indicated, such as hydrocyanic acid, aconite, hyoscyamus, opium in small doses, etc. It is considered by some writers injudicious to resort to digitalis for this purpose; but I can see no just grounds for apprehending evil from the use of this remedy, if proper caution be observed. Here, as in other instances in which it is desirable to reduce the frequency of the heart's action, it is a remedy of great value.

[1] *e. g.* Bellingham, op. cit.

Certain remedies are recommended with a view to induce a condition of the blood favoring the coagulation of the fibrin. Dr. Hope regarded the acetate of lead as useful in this way; and Dr. Walshe attaches some value to gallic or tannic acid. Theoretically, remedies are indicated which produce this effect, provided it be not counterbalanced by other consequences. It is, however, doubtful whether these or other known remedies produce this effect; and, moreover, it is to be considered that ordinary coagulation of blood within the sac, so far from being desirable, would be likely to give rise to serious results. Coagulation is conservative and curative only when it takes place at the bottom of the sac, leading to the formation of layers of fibrin, which become adherent first to the walls of the sac, and successively to each other, until the cavity is more or less filled with the solid deposit.

The various symptomatic and remote effects of thoracic aneurisms, such as pain, cerebral congestion, labored respiration, cough, dysphagia, etc., will, of course, claim palliative measures of treatment, which are to be adapted to the particular circumstances of individual cases, and need not be here considered.

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